Department of Psychology and Logopedics
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# Neurocognitive functioning and psychiatric symptoms in children and adolescents with higher functioning autism spectrum disorders

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ACADEMIC DISSERTATION

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# Abstract

Autism spectrum disorders (ASD) are characterized by social interaction and communication difficulties, and by restrictive, repetitive and stereotyped patterns of behavior. In many ways, ASD is a highly heterogeneous disorder, and it is not known why some individuals with ASD end up having a poor outcome while others may cope well. Understanding of associated neurocognitive and psychiatric factors in ASD is crucial for enabling planning suitable follow-ups, as well as planning effective interventions. This thesis investigates neurocognitive functioning and psychiatric symptoms comprehensively in children and adolescents with higher functioning ASD (HF-ASD) in four studies.

In Studies I and II, the neurocognitive functioning of children and adolescents with HF-ASD were compared with that of typically developing (TD) children and adolescents. In Study III, psychiatric symptoms of children and adolescents with HF-ASD were compared to that of TD children and adolescents, and to the reported prevalence rates of psychiatric symptoms by Ford, Goodman, and Melzer (2003). In Study IV, children and adolescents with HF-ASD were divided into three groups based on the level of symptoms of sluggish cognitive tempo (SCT): the ASD+High SCT group, the ASD+Medium SCT group, and the ASD+Low SCT group. The groups were compared on social skills and academic functioning, internalizing and externalizing psychiatric symptoms and processing speed.

The present results showed that children and adolescents with HF-ASD had strengths in verbal reasoning skills and weaknesses in attention and executive functions (EF), facial recognition memory, and visuomotor functions. Overall, the neurocognitive deficits in children and adolescents with HF-ASD at the group-level were mild. In contrast, children and adolescents with HF-ASD had high rates of co-occurring psychiatric symptoms. Particularly anxiety and depression, attention deficit hyperactivity disorder and tic disorders were frequent in these individuals. Finally, the HF-ASD+High SCT group and the HF-ASD+Medium SCT group had more pronounced social difficulties than the HF-ASD+Low SCT group. Additionally, the HF-ASD+High SCT group had a higher rate of symptoms of anxiety and depression compared to the HF-ASD+Low SCT group.

To conclude, these results suggest that children and adolescents with HF-ASD are characterized by mild neurocognitive deficits in single clinical neuropsychological subtests. High co-occurrence of psychiatric symptoms in children and adolescents with HF-ASD emphasize the importance of evaluating psychiatric symptoms systematically in HF-ASD. Results on SCT symptoms in HF-ASD indicate that individuals with HF-ASD and high levels of SCT symptoms may be at risk for pronounced social difficulties and internalizing psychiatric symptoms. Therefore, identifying individuals with HF-ASD and with symptoms of SCT would be important for planning systematical follow-ups and preventive support for these individuals.

# Tiivistelmä

Autismikirjon häiriöiden (autism spectrum disorders, ASD) ydinoireita ovat sosiaalisen vuorovaikutuksen ja kommunikaation vaikeudet sekä rajoittavat, toistavat ja kaavamaiset käyttäytymispiirteet. ASD on heterogeeninen häiriö eikä tiedetä, että miksi osalla ASD-diagnoosin saaneista henkilöistä on vaikeuksia selviytyä yhteiskunnan vaatimuksista, kun taas osa heistä selviytyy hyvin. Siksi neurokognitiivisten ja psykiatristen tekijöiden tunteminen ASD:ssa on edellytys sopivan seurannan ja tehokkaiden tukitoimien suunnittelun mahdollistamiseksi. Tämän väitöskirjan neljässä osajulkaisussa tutkittiin neurokognitiivista suoriutumista ja psykiatrisia oireita lapsilla ja nuorilla, joilla oli diagnosoitu ASD ja joiden älykkyysosamäärä oli ≥ 70 (HF-ASD).

Ensimmäisessä ja toisessa osajulkaisussa HF-ASD-diagnoosin saaneiden lasten ja nuorten neurokognitiivista suoriutumista verrattiin tyypillisesti kehittyneiden lasten ja nuorten neurokognitiiviseen suoriutumiseen. Kolmannessa osajulkaisussa HF-ASD-diagnoosin saaneiden lasten ja nuorten psykiatrisia oireita verrattiin tyypillisesti kehittyneiden lasten ja nuorten psykiatrisiin oireisiin sekä Fordin, Goodmanin ja Meltzerin (2003) raportoimiin esiintyvyyslukuihin. Neljännessä osajulkaisussa HF-ASD-diagnoosin saaneet lapset ja nuoret jaettiin kolmeen ryhmään sen perusteella missä määrin (paljon, kohtalaisesti, vähän) heillä oli verkkaiseen kognitiiviseen tahtiin (sluggish cognitive tempo, SCT) viittaavia oireita. Näitä kolmea ryhmää verrattiin keskenään sosiaalisten taitojen, akateemisen suoriutumisen, internalisoivien ja eksternalisoivien psykiatristen oireiden sekä prosessoinnin nopeuden osalta.

Tulokset osoittivat, että HF-ASD-diagnoosin saaneilla lapsilla sekä nuorilla on vahvuuksia kielellisessä päättelysuoriutumisessa ja vaikeuksia tarkkaavuuden säätelyssä sekä toiminnanohjauksessa, kasvojen tunnistamisessa ja silmän- sekä käden yhteistyössä. Neurokognitiiviset vaikeudet HF-ASD-diagnoosin saaneilla lapsilla ja nuorilla näyttäytyivät ryhmätasolla lieväasteisina. Sen sijaan HF-ASD-diagnoosin saaneilla lapsilla ja nuorilla oli tässä tutkimuksessa paljon psykiatrisia oireita. Näitä olivat ahdistuneisuus- sekä masennusoireet, tarkkaavaisuus- ja ylivilkkaushäiriön oireet sekä tic-oireet. Viimeiseksi tulokset osoittivat, että HF-ASD diagnoosin saaneilla lapsilla ja nuorilla, joilla oli paljon tai kohtalaisesti SCT oireita, oli vaikea-asteisempia sosiaalisen vuorovaikutuksen hankaluuksia kuin HF-ASD-diagnoosin saaneilla lapsilla ja nuorilla, joilla oli vähän SCT-oireita. Lisäksi tulokset osoittivat, että HF-ASD-diagnoosin saaneilla lapsilla ja nuorilla, joilla oli paljon SCT-oireita, oli enemmän ahdistus- ja masennusoireita kuin ASD-diagnoosin saaneilla lapsilla ja nuorilla, joilla oli vähän SCT-oireita.

Yhteenvetona todettakoon, että tutkimuksen tulokset viittaavat siihen, että HF-ASD-diagnoosin saaneilla lapsilla neurokognitiiviset vaikeudet ovat lieväasteisia ja tulevat esiin yksittäisissä kliinisissä neuropsykologisissa osatesteissä. HF-ASD-diagnoosin saaneilla lapsilla ja nuorilla on paljon psykiatrisia oireita, minkä vuoksi näiden oireiden arvioiminen on keskeistä. Tulokset SCT-oireista HF-ASD:ssä viittaavat siihen, että niillä lapsilla ja nuorilla, joilla on HF-ASD diagnoosin lisäksi paljon SCT-oireita, voi olla riski vaikea-asteisempiin sosiaalisiin vaikeuksiin sekä internalisoiviin psykiatrisiin oireisiin. Siten SCT-oireiden tunnistaminen HF-ASD:ssä on tärkeää, jotta näille lapsille ja nuorille voidaan suunnitella systemaattinen seuranta sekä järjestää ennaltaehkäiseviä tukitoimia.

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# List of original publications

This thesis is based on the following publications:

I Barron-Linnankoski, S., Reinvall, O., Lahervuori, A., Voutilainen, A., Lahti-Nuuttila, P., & Korkman, M. (2015). Neurocognitive performance of children with higher functioning autism spectrum disorders on the NEPSY-II. *Child Neuropsychology, 21*(1)*,* 55-77. doi:10.1080/09297049.2013.873781

II Reinvall, O., Voutilainen, A., Kujala, T., & Korkman, M. (2013). Neurocognitive functioning in adolescents with higher functioning autism spectrum disorder. *Journal of Autism and Developmental Disorders, 43* (6), 1367-1379. doi:10.1007/s10803-012-1692-8

III Reinvall, O., Moisio, A.-L., Lahti-Nuuttila, P., Voutilainen, A., Laasonen, M., & Kujala, T. (2016). Psychiatric symptoms in children and adolescents with higher functioning autism spectrum disorders on the Development and Well-Being Assessment. *Research in Autism Spectrum Disorders, 25,* 47-57. doi:10.1016/j.rasd.2016.01.009

IV Reinvall, O., Kujala, T., Voutilainen, A., Moisio, A.-L., Lahti-Nuuttila, P., & Laasonen, M. (2017). Sluggish cognitive tempo in children and adolescents with higher functioning autism spectrum disorders: Social impairments and internalizing symptoms. *Scandinavian Journal of Psychology, 58*(5), 389-399. doi: 10.1111/sjop.12379

The publications are referred to in the text by their roman numerals.

## Abbreviations

| | |
|---|---|
| AS | Asperger syndrome |
| ASD | autism spectrum disorders |
| ADI-R | Autism Diagnostic Interview-Revised |
| DAWBA | Development and Well-Being Assessment |
| DSM-IV | Diagnostic and Statistical Manual of Mental Disorders, 4th edition |
| DSM-5 | Diagnostic and Statistical Manual of Mental Disorders, 5th edition |
| EF | executive functions |
| FSIQ | full scale intelligence quotient |
| FTF | Five-to-Fifteen Questionnaire |
| HFA | high functioning autism |
| HF-ASD | higher functioning autism spectrum disorder |
| ICD-10 | International Classification of Diseases, 10th edition |
| LFA | low functioning autism |
| NEPSY | a developmental neuropsychological assessment |
| NEPSY-II | a developmental neuropsychological assessment, second edition |
| PIQ | performance intelligence quotient |
| SCT | sluggish cognitive tempo |
| TD | typically developing |
| VIQ | verbal intelligence quotient |
| WISC-III | Wechsler Intelligence Scale for Children, 3rd edition |
| ToM | theory of mind |

# 1 Introduction

Social interaction difficulties and communication impairments, as well as restricted and repetitive behaviors, are the three core symptom domains defining autism spectrum disorders (ASD) (American Psychiatric Association, 1994; World Health Organization, 1993). No single cause of ASD has been identified. Instead, ASD appears to be resulting from multiple factors and their interaction (Chen, Peñagarikano, Belgard, Swarup, & Geschwind, 2015; Lord & Bishop, 2015; Miles, 2011). Genetic factors have a significant contribution to the etiology of ASD as indicated by the associations between ASD and genetic disorders and by the results of twin and family studies (Chen et al., 2015). Tens or probably hundreds of genetic or genomic defects may contribute to ASD emphasizing the complexity of genetic etiology in this disorder (Betancur, 2011). Neuropathological abnormalities indicate deficits in the development of forming long-range neural connections and excess forming of short-range connections (Geschwind & Levitt, 2007). Disarranged connectivity of frontoparietal, frontotemporal, frontostriatal, and interhemispheric circuits in ASD has been found, as well as abnormalities in the cerebellum (Chen et al., 2015). In addition to genetic and neural level causes of ASD, environmental factors contributing to the etiology of ASD have been identified including pre-, peri- and neonatal factors (for meta-analyses see, Gardener, Spiegelman, & Buka, 2009; Gardener, Spiegelman, & Buka, 2011). The strongest prenatal risk factors for autism include advanced parental age, being born first versus being born third or later, maternal bleeding during pregnancy, maternal gestational diabetes, having a mother who was born abroad and maternal prenatal medication (for a meta-analysis see, Gardener et al., 2009). Several peri- and neonatal risk factors for autism have been found although it is not known whether these are resulting from prenatal complications (for a meta-analysis see, Gardener et al., 2011).

Although previously thought as a rare disorder, the prevalence rates of ASD have been rising in the past two decades (Lord & Bishop, 2015). Sixty-two out of 10 000 individuals worldwide were estimated to have an ASD based on a systematic review regarding epidemiological surveys (Elsabbagh et al., 2012). In Finland, the annual incidence rate of ASD in children under ten years was reported to be 53.7 individuals out of 10 000 individuals according to a population-based cohort study conducted with children who were born between 1996 and 1998 (Hinkka-Yli-Salomäki et al., 2014). Males are more likely to have an ASD than females with the mean male:female ratio being 4.2:1 and ranging from 1.4:1 to 16:1 (Fombonne, 2009). In Finland, the male:female ratio was reported to be 3.5:1 (Hinkka-Yli-Salomäki et al., 2014). The overall outcome consisting independent living, educational/occupational and social functioning in individuals with HF-ASD (i.e., Asperger syndrome/high functioning autism) across two different studies was estimated to be good in 12% and 27% of the individuals, fair in 70% and 75% of the subjects and poor in 3% and 12% of the participants (Steinhausen, Mohr Jensen, & Lauritsen, 2016). Research indicates that neurocognitive factors such as intellectual functioning and language abilities are two of the strongest predictors of adult outcome in individuals with ASD (for a review see, Howlin & Magiati, 2017). Additionally, the severity of co-occurring psychiatric symptoms has been found to be predictive of school

performance and social functioning in ASD (Gadow, DeVincent, & Schneider, 2008). Therefore, understanding of associated neurocognitive and psychiatric factors in ASD forms the basis for enabling planning suitable follow-ups, as well as planning effective interventions to enable better outcome in ASD. A growing number of studies have been conducted in recent years regarding ASD; however, comprehensive studies on neurocognitive functioning and psychiatric symptoms in ASD applying clinically relevant methods are scarce. Additionally, the heterogeneity in samples and methods makes it difficult to conclude the results.

## 1.1 Higher functioning ASD

Asperger syndrome belongs to the continuum of ASD. The first descriptions of ASD date back to Leo Kanner's publication in 1943 "Autistic disturbances of affective contact" and Hans Asperger's publication in 1944 "Die „Autistischen Psychopathen" im Kindesalter" (Wing, 1981). However, only after Lorna Wing's publication "Asperger's syndrome: A clinical account" in 1981, the term Asperger syndrome started to become more into use (Frith, 2004). In 1993 and 1994, the diagnosis of Asperger syndrome was incorporated to International Classification of Diseases (10th ed.; ICD-10; World Health Organization, 1993) and Diagnostic and Statistical Manual of Mental Disorders (4th ed.; DSM-IV; American Psychiatric Association, 1994) diagnostic classifications. The main distinction between Asperger syndrome diagnosis and autism diagnosis in DSM-IV (American Psychiatric Association, 1994) and ICD-10 (World Health Organization, 1993) is that in Asperger syndrome the early development of language and cognition should not generally be delayed whereas in autism these delays are allowed (Frith, 2004; Lord, Cook, Leventhal, & Amaral, 2000). In individuals with autism, intelligence quotients (IQ) can vary from intellectual disability to average range or above (Chiang, Tsai, Cheung, Brown, & Li, 2014; Lord et al., 2000). This has led to the need to describe individuals with autism more specifically beyond DSM-IV (American Psychiatric Association, 1994) and ICD-10 (World Health Organization, 1993) diagnoses. In research, the term low functioning autism (LFA) is often used to characterize people with autism and an IQ below 70 (Bölte & Poustka, 2002; McGovern & Sigman, 2005; Preissler, 2008), while individuals with autism and IQ above 70 have been described to as having high functioning autism (HFA). A considerable amount of research has focused on investigating whether Asperger syndrome is a distinct disorder from HFA. Several genetic, neurophysiological, cognitive, and behavioral studies indicate that they are not essentially distinctive disorders, but instead belong to the continuum of autistic disorders differing with respect to the degree of severity (Frith, 2004).

In 2013, the diagnosis of Asperger syndrome was removed from Diagnostic and Statistical Manual of Mental Disorders (5th ed., DSM-5; American Psychiatric Association, 2013). At the moment, only single diagnosis of ASD is used in DSM-5 (American Psychiatric Association, 2013) to describe previously separate diagnoses of DSM-IV (American Psychiatric Association, 1994) including autism, Asperger syndrome, pervasive developmental disorders not otherwise specified, and disintegrative disorders.

According to DSM-5 (American Psychiatric Association, 2013), individuals with clinically well-informed DSM-IV (American Psychiatric Association, 1994) diagnoses of Asperger syndrome can be assumed to fulfill the DSM-5 (American Psychiatric Association, 2013) diagnostic criteria of ASD and need not to be re-diagnosed. The current thesis concentrates on examining children and adolescents who were diagnosed to have Asperger syndrome. The term HF-ASD is used throughout the thesis to describe children and adolescents with Asperger syndrome and/or HFA to reflect the changes in DSM-5 (American Psychiatric Association, 2013) and research literature when appropriate.

## 1.2 Neurocognitive theories of ASD

The theory of executive dysfunction, the account on theory of mind (ToM) deficits, and the weak central coherence (WCC) theory are the three main neurocognitive theories used to account the core impairments in ASD (Frith, 1997; Hill & Frith, 2003; Rajendran & Mitchell, 2007). The executive dysfunction theory posits that impairments in frontal lobes in ASD are linked to executive function (EF) deficits, which could explain symptoms of restricted and repetitive patterns of behavior in ASD (Hill, 2004). Executive functions refer to a wide range of goal-directed cognitive and adaptive skills that are needed in situations demanding attention, concentration and effort (Diamond, 2013). Thus, EFs are critical in new situations, performing novel tasks and in situations with sudden changes, which require flexible problem-solving. Conversely, EFs are less needed in familiar situations or in tasks, which are structured, well-learned and/or automatized. Currently, inhibition, working memory, and cognitive flexibility or set-shifting are generally agreed to form the three main EFs (Diamond, 2013; Miyake et al., 2000). More advanced EFs, such as planning, problem-solving, and reasoning are hypothesized to build upon these three core EFs (Diamond, 2016; Diamond, 2013). At the behavioral level, characteristics fitting to the executive dysfunction theory in individuals with ASD are their ability to perform familiar tasks, and their strong tendency to routines, rituals, and repetitive actions while at the same time having poor management of changes, rigidity, and impaired coping in daily life with high EF demands is frequent (Hill, 2004). Overlap with other clinical disorders (i.e., attention deficit hyperactivity disorder (ADHD), non-specific hypotheses of which EFs are dysfunctional in ASD, and mixed research results regarding EFs in ASD are the weaknesses of this theory (Hill & Frith, 2003).

The ToM deficits account posits that individuals with ASD have an impairment in ToM which is linked to difficulties in pretend play and to deficits in social skills (Baron-Cohen, Leslie, & Frith, 1985). The concept of ToM was used by Premack and Woodruff (1978), and it refers to the ability to impute mental states to others, as well as to oneself. Mental states include an understanding of what other people might be believing, wanting, knowing, and feeling (Baron-Cohen et al., 1985). The ToM is crucial for social communication and interaction since ToM enables an individual to understand and predict other people's actions (Baron-Cohen et al., 1985; Frith, Morton, & Leslie, 1991; Hill & Frith, 2003). The ToM account has been linked with at least part of the social interaction

difficulties in ASD including both withdrawal and indiscriminate social approach (Baron-Cohen et al., 1985; Frith et al., 1991). One of the first studies to test the ToM account in autism was the study of Barron-Cohen et al. (1985). They found that children with autism were impaired in a ToM task named "Sally-Ann test". Only 20% of children with autism passed this test while the comparable percentage of children with Down syndrome was 86% although children with Down syndrome had lower non-verbal and verbal mental age than children with autism. A limitation of the ToM account is the inability to explain the restricted and repetitive patterns of behavior in ASD (Happé & Ronald, 2008).

The theory of WCC, first introduced by Frith (1989), posits that individuals with ASD have an impaired central coherence, that is, deficient ability to process information contextually or globally and tendency to process information in a detailed-focused way or locally. This theory has been modified afterward. Currently, WCC is not considered as a core deficit in ASD but instead as a secondary outcome of possibly superior locally focused processing style (Happé, Booth, Charlton, & Hughes, 2006; Hill & Frith, 2003). According to this view, individuals with ASD can overcome their locally focused cognitive bias if needed and are capable of process information also globally (Happé & Frith, 2006). The WCC theory has been used to explain both strengths and weaknesses in ASD. Strengths accounted by the WCC in ASD include super-acute perception and uneven intellectual profile with peaks in performances requiring detail-focused attention (Happé & Frith, 2006; Hill & Frith, 2003). Weaknesses explained by the WCC in ASD consist of difficulty accepting minor changes in the environment and impaired generalization (Happé & Frith, 2006). Additionally, the WCC has been hypothesized to influence face and facial emotion recognition (Happé & Frith, 2006). A limitation of the WCC account is that not much is known about the neural level of WCC (Hill & Frith, 2003).

## 1.3 Neurocognitive functioning in HF-ASD

Only a few studies have assessed neurocognitive functioning comprehensively in children and adolescents with HF-ASD in comparison to typically developing (TD) children and adolescents. The majority of neurocognitive studies in HF-ASD have focused on examining intellectual performance, EFs, and social cognition. Sometimes a higher Verbal Intelligence Quotient (VIQ) compared to Performance Intelligence Quotient (PIQ) has been found in children and adolescents with Asperger syndrome in studies examining the VIQ and PIQ discrepancy within the Asperger syndrome group (Ghaziuddin & Mountain-Kimchi, 2004; Ozonoff, South, & Miller, 2000). In other studies, such discrepancies have not been reported (Manjiviona & Prior, 1999; Spek, Scholte, & van Bercelaer-Onnes, 2008).

In a more detailed level, children and adolescents with Asperger syndrome have typically the highest scaled scores in the Information (mean scores ranging from 10.7 to 13.3 between studies) and Similarities (mean scores ranging from 10.2 to 13.1 between studies) VIQ subtests of the Wechsler Intelligence Scales (Barnhill, Hagiwara, Myles, & Simpson, 2000; Ghaziuddin & Mountain-Kimchi, 2004; Manjiviona & Prior, 1999;

Noterdaeme, Wriedt, & Höhne, 2010; Planche & Lemonnier, 2012). The lowest scaled scores of the Wechsler Intelligence Scales VIQ subtests in children and adolescents with Asperger syndrome are usually reported in the Arithmetic (mean scores ranging from 8.9 to 11.4 between studies) and Comprehension (mean scores ranging from 9.1 to 9.5 between studies) (Barnhill et al., 2000; Ghaziuddin & Mountain-Kimchi, 2004; Manjiviona & Prior, 1999; Noterdaeme et al., 2010; Planche & Lemonnier, 2012). Regarding PIQ subtests of the Wechsler Intelligence Scales, the highest scaled scores are found typically in the Block Design (mean scores ranging from 11.0 to 12.6 between studies) and Picture Completion (mean scores ranging from 9.9 to 13.5 between studies) while the lowest scaled scores are usually reported in the Coding (mean scores ranging from 4.0 to 8.1 between studies) and Picture Arrangement (mean scores ranging from 8.1 to 10.6 between studies) (Barnhill et al., 2000; Ghaziuddin & Mountain-Kimchi, 2004; Manjiviona & Prior, 1999; Noterdaeme et al., 2010; Planche & Lemonnier, 2012). It should be noted that all of these scaled scores correspond the average range performance with the exception of the Coding subtest of the WISC-III in which the scaled scores have been below 8 (Barnhill et al., 2000; Ghaziuddin & Mountain-Kimchi, 2004; Manjiviona & Prior, 1999; Noterdaeme et al., 2010; Planche & Lemonnier, 2012).

With respect to EFs, the most consistent findings in individuals with HF-ASD relate to deficits in set-shifting or cognitive flexibility, planning, and response selection/monitoring (Happé et al., 2006; Nydén, Gillberg, Hjelmquist, & Heiman, 1999; Semrud-Clikeman, Walkowiak, Wilkinson, & Butcher, 2010). From a developmental perspective, it is not clear whether EF impairments reported in childhood persist to adolescence and adulthood in individuals with HF-ASD. Happé et al. (2006) reported in their cross-sectional study that younger individuals with HF-ASD performed significantly lower than younger TD participants in response selection/monitoring while older individuals with HF-ASD performed as well as older TD individuals in response selection/monitoring and in several other EF measures. Overall, comparing EF studies conducted in individuals with ASD is difficult due to the wide variability of EF definitions, tasks used to assess EF, and high heterogeneity between the samples regarding background variables in different studies.

Social cognition refers to a wide variety of processes enabling individuals to interact with each other (Frith & Frith, 2007). The core processes of social cognition have been suggested to include agent-identification, affiliation, emotion processing, emotional empathy, self-processing, in-group/out-group categorization, social hierarchy mapping, mental state attribution, social policing, and individual's information store (Happé & Frith, 2014). The majority of neurocognitive studies in individuals with HF-ASD have focused on assessing ToM abilities (part of the mental state attribution process), and face (part of the agent-identification process) and emotion expression recognition (part of the emotion processing process). The ToM is most often assessed with first-order and second-order false belief tasks. First-order false belief tasks examine the skill to infer one person's mental state and that in the same situation different people can have different thoughts (Baron-Cohen, 2001). Second-order false belief tasks investigate the skill to infer what another individual thinks a second individual knows and/or beliefs (Baron-Cohen, 2001). Some studies indicate that individuals with HF-ASD pass the first- and/or second-order ToM tasks (Bowler, 1992; Dahlgren & Trillingsgaard, 1996). In advanced theory of mind

tasks, such as the Strange Stories, the Eyes Task and the Stories from Everyday Life task, impairments in ToM skills have been found in children and adolescents with HF-ASD (Barnhill et al., 2000; Happé, 1994; Kaland et al., 2002; Kaland, Smith, & Mortensen, 2008).

Face processing deficits have been found in individuals with HF-ASD although the research results have been mixed (Behrmann et al., 2006; Best, Minshew, & Strauss, 2010; Klin et al., 1999; Kuusikko-Gauffin et al., 2011). Kuusikko-Gauffin et al. (2011) found that younger individuals with HF-ASD performed at a lower level compared to TD group while no significant differences were found regarding older individuals with HF-ASD and TD group indicating age-related improvements concerning face memory in HF-ASD. In contrast, no significant group differences in face memory were reported by O'Hearn, Schroer, Minshew, and Luna (2010) in children and adolescents with versus without HF-ASD whereas face memory was significantly deficient in adults with HF-ASD compared to TD adults.

Emotion expression recognition is most often assessed with tasks requiring the identification of six basic emotion expressions which are anger, joy, fear, disgust, sadness, and surprise. In addition to these six basic emotion expressions, a neutral expression is often included in the studies. Recognizing basic emotion expressions have been found to be deficient in children and adolescents with HF-ASD in some studies (Krebs et al., 2011; Kuusikko et al., 2009; Lindner & Rosen, 2006; Rump, Giovannelli, Minshew, & Strauss, 2009), while in other studies such impairments have not been reported (Jones et al., 2011; Tracy, Robins, Schriber, & Solomon, 2011). For studies reporting deficits in this area, it is not clear which of the basic emotion expressions are deficient in individuals with HF-ASD. Sadness and fear are the most often found impairments but deficits in other basic emotion expressions have been reported as well (Ashwin, Wheelwright, & Baron-Cohen, 2006; Boraston, Blakemore, Chilvers, & Skuse, 2007; Corden, Chilvers, & Skuse, 2008; Pelphrey et al., 2002; Wallace, Coleman, & Bailey, 2008).

Fewer studies have assessed language, memory and learning, visuospatial and sensorimotor functions in HF-ASD. Regarding language, the majority of studies have focused on investigating the social use of language in these individuals. The few studies conducted in children with HF-ASD regarding basic language skills indicate difficulties in expressive and receptive language. Szatmari, Archer, Fisman, Streiner, and Wilson (1995) found that children with Asperger syndrome scored below normal on tasks of expressive and receptive language abilities. In line with these results, expressive language impairments were found in 33% and receptive language deficits in 39% of children and adolescents with Asperger syndrome in a study by Noterdaeme et al. (2010). Additionally, Saalasti et al. (2008) reported receptive language deficits in children with Asperger syndrome compared to TD children on a task investigating comprehension of verbal instructions. Only a few studies have been conducted concerning memory and learning in children and adolescents with HF-ASD beyond working memory and face memory. Children with HF-ASD have been found to produce less coherent stories compared to TD children in story memory tasks (Diehl, Bennetto, & Young, 2006; Losh & Gordon, 2014). Concerning adolescents with HF-ASD, Minshew and Goldstein (2001) found that individuals with HF-ASD (age range 12-40; mean age 22.3) had deficits in list learning,

and story recall compared to the TD group. Additionally, Minshew and Goldstein (2001) reported that recall of a complex figure was impaired in individuals with HF-ASD compared to TD adolescents and young adults.

Research concerning visuospatial abilities in children and adolescents with HF-ASD is scarce. In children with HF-ASD Klin, Volkmar, Sparrow, Cicchetti, and Rourke (1995) reported impairments in visuospatial skills, while in other studies such differences have not been found between children and adolescents with HF-ASD and TD children and adolescents (Edgin & Pennington, 2005; Ham, Corley, Rajendran, Carletta, & Swanson, 2008; Minshew, Goldstein, & Siegel, 1997; Semrud-Clikeman, Walkowiak, Wilkinson, & Christopher, 2010). Regarding sensorimotor abilities, fine-motor slowness, impairments in visuomotor integration, and deficits in imitating finger and hand positions have been reported in children and adolescents with HF-ASD (Ham et al., 2008; Klin et al., 1995; Manjiviona & Prior, 1995; Volker et al., 2010).

To summarize, results of the neurocognitive studies in children and adolescents with HF-ASD have been mixed, a comparison between the studies is difficult, and generalizations to clinical practice is challenging due to the heterogeneity concerning 1) age in samples, 2), diagnostic and control groups used, and 3) assessment methods. The majority of neurocognitive studies in HF-ASD have focused on examining intellectual performance, EFs, and social cognition. Less is known about language, memory and learning, visuospatial functions, and sensorimotor abilities in HF-ASD. One beneficial approach for studying neurocognitive functioning in ASD would be to increase the homogeneity within the sample concerning variables that may have an obscuring effect on the results. Based on the research reviewed above, potential variables affecting the results are developmental delays in language and cognitive functioning, age, and IQ. Additionally, applying a standardized assessment measure covering comprehensively neurocognitive functions would enable the use of same norms across different domains of functioning.

## 1.4 Psychiatric symptoms in HF-ASD

A large body of evidence indicate that the majority of individuals with ASD have one or multiple co-occurring psychiatric disorders (Ghaziuddin, Weidmer-Mikhail, & Ghaziuddin, 1998; Gjevik, Eldevik, Fjæran-Granum, & Sponheim, 2011; Mattila et al., 2010; Simonoff et al., 2008; Tonge, Brereton, Gray, & Einfeld, 1999). The rates of psychiatric disorders in individuals with ASD have varied from 65% to 74% between studies that have assessed psychiatric symptoms with semi-structured or structured diagnostic interviews (Ghaziuddin et al., 1998; Gjevik et al., 2011; Mattila et al., 2010; Simonoff et al., 2008). Neurobiological and environmental factors, as well as factors relating to ASD, have been hypothesized as predisposing individuals with HF-ASD to psychiatric symptoms (Mannion, Brahm, & Leader, 2014; Tantam, 2000; White & Roberson-Nay, 2009). The association between IQ and psychiatric symptoms in individuals with ASD is not clear. No relationships between IQ and psychiatric symptoms have been reported in interview-based studies (Gjevik et al., 2011; Simonoff et al., 2008).

However, significant associations between IQ and emotional disorders (i.e., anxiety and depression) have been found in questionnaire-based studies (Gadow, Guttmann-Steinmetz, Rieffe, & DeVincent, 2012; Sukhodolsky et al., 2008; Weisbrot, Gadow, DeVincent, & Pomeroy, 2005). Particularly, higher IQs have been related with higher scores of anxiety (Sukhodolsky et al., 2008; Weisbrot et al., 2005) and depressive symptoms in individuals with ASD (Gadow et al., 2012; Vickerstaff, Heriot, Wong, Lopes, & Dossetor, 2007). Researchers have suggested that individuals with HF-ASD may be more aware of being different from their peers and of their social difficulties and therefore they are at elevated risk of having psychiatric symptoms (Gadow et al., 2012; Rieffe et al., 2011; Vickerstaff et al., 2007).

Anxiety disorders are frequent in children and adolescents with HF-ASD (Mattila et al., 2010; Mazefsky et al., 2012; Mukaddes & Fateh, 2010; Mukaddes, Hergüner, & Tanidir, 2010). Especially specific phobias and obsessive-compulsive disorder (OCD) are common in ASD (for a meta-analysis see, van Steensel, Bögels, & Perrin, 2011). Research regarding post-traumatic stress disorder (PTSD) symptoms in children and adolescents with HF-ASD is scarce. The percentages of PTSD ranged from 0% to 18% in studies including participants with LFA (de Bruin, Ferdinand, Meester, de Nijs, & Verheij, 2007; Mehtar & Mukaddes, 2011; van Steensel et al., 2011). Elevated risk for traumatic events and for developing a PTSD can be higher in individuals with ASD due to difficulties in emotion regulation and social cognition (Kerns, Newschaffer, & Berkowitz, 2015). Additionally, in TD children and adolescents being bullied has been found to be related with PTSD symptoms (Guzzo, Pace, Cascio, Craparo, & Schimmenti, 2014; Idsoe, Dyregrov, & Idsoe, 2012). Since children and adolescents with ASD often experience long-lasting and frequent bullying (Cappadocia, Weiss, & Pepler, 2012), which can be traumatic, assessing PTSD in ASD would be beneficial.

Depression in adults with HF-ASD is a common co-occurring disorder, with the rates varying from 43% to 52% in interview-based studies (Ghaziuddin et al., 1998; Hofvander et al., 2009; Sterling, Dawson, Estes, & Greenson, 2008). Much lower rates of depression have been reported in children with HF-ASD in interview-based studies. In these studies, the rates of major depression have ranged from 6% to 13% (Mattila et al., 2010; Mukaddes & Fateh, 2010).

Behavioral disorders often co-occur in HF-ASD (Gjevik et al., 2011; Green, Gilchrist, Burton, & Cox, 2000; Klin, Pauls, Schultz, & Volkmar, 2005; Mattila et al., 2010). High rates of ADHD have been found in several studies in individuals with HF-ASD (Ghaziuddin et al., 1998; Green et al., 2000; Lee & Ousley, 2006; Mattila et al., 2010; Mukaddes et al., 2010). The rates of ADHD combined (ADHD-C, i.e., both inattention and hyperactivity-impulsivity) and ADHD inattentive (ADHD-I) subtype have varied in these studies, while the rates of ADHD hyperactive-impulsive subtype (ADHD-HI) have been consistently low in children and adolescents with HF-ASD (Lee & Ousley, 2006; Mattila et al., 2010; Mukaddes et al., 2010). Not much is known regarding co-occurring oppositional defiant disorder (ODD) and conduct disorder (CD) in HF-ASD, and the findings have been varied (Green et al., 2000; Mattila et al., 2010; Mukaddes & Fateh, 2010; Mukaddes et al., 2010).

Tic disorders have been found in individuals with ASD (Canitano & Vivanti, 2007; Gjevik et al., 2011; Hofvander et al., 2009). However, only a few studies have examined the rates of particular types of tic disorders, such as chronic tic disorder, Tourette syndrome (TS), or tic disorder not otherwise specified (NOS) in children and adolescents with HF-ASD (Mattila et al., 2010; Mukaddes & Fateh, 2010). Concerning eating disorders, in adolescents and adults with anorexia nervosa, the rate of co-occurring ASD has been reported to vary from 5% to 23% (Attwood, 2007; Gillberg & Råstam, 1992; Pooni, Ninteman, Bryant-Waugh, Nicholls, & Mandy, 2012; Råstam, Gillberg, & Wentz, 2003). However, less is known about anorexia nervosa and bulimia nervosa in HF-ASD, especially regarding children and adolescents. Other eating disorders, including pica, and eating-related difficulties, such as limited food repertoire seem to be common in ASD (Ahearn, Castine, Nault, & Green, 2001; Bandini et al., 2010; Emond, Emmett, Steer, & Golding, 2010).

To summarize, a growing number of studies regarding psychiatric symptoms in HF-ASD have been conducted. Some factors, however, pose challenges to the interpretation of the results. First, only limited number of studies has investigated psychiatric symptoms comprehensively across different domains and in detail. Thus, rates of PTSD, depression, tic disorders, or eating disorders are understudied in children and adolescents with HF-ASD. Second, including a TD control group when examining psychiatric symptoms in individuals with HF-ASD would be important since background variables, such as age, gender, and parental education have been found to be related with psychiatric symptoms in population-based studies (Ford, Goodman, & Meltzer, 2003; Frigerio et al., 2009; Heiervang et al., 2007). Previous studies on psychiatric symptoms in HF-ASD have not used a TD control group (Ghaziuddin et al., 1998; Gjevik et al., 2011; Mattila et al., 2010; Mazefsky et al., 2012; Mukaddes & Fateh, 2010; Mukaddes et al., 2010; Simonoff et al., 2008). Third, some studies have used mixed samples consisting of individuals with LFA and HF-ASD (Gjevik et al., 2011; Simonoff et al., 2008). Since IQ may affect on the psychiatric symptoms in individuals with ASD, examining psychiatric symptoms separately in these individuals would be recommended.

## 1.5 Sluggish cognitive tempo and HF-ASD

The construct of sluggish cognitive tempo (SCT) dates back to 1980's when in factor-analytic studies of ADHD (at that time named attention deficit disorder; ADD) a third factor named "Sluggish Tempo" was found in addition to inattention and hyperactivity-impulsivity factors. The "Sluggish Tempo" factor consisted of symptoms of hypoactivity or inattention separate of ADD symptoms in DSM-III (Lahey et al., 1988). At the moment, several studies indicate that SCT symptoms are distinct from ADHD symptoms. Factor-analytic studies and studies on background variables support this view. The majority of factor-analytic studies (21 out of 23) have found the SCT symptoms loading on a factor or factors separate from ADHD-I and ADHD-HI factors (for a meta-analysis and critical review see, Becker, Leopold et al., 2016a). Studies on background variables indicate that symptoms of SCT have different associations with age, ethnicity, and sex

than ADHD symptoms (Barkley, 2014). It should be noted, however, that overlap between ADHD and SCT exists. The symptom dimensions of SCT and ADHD-I correlate moderately, and weak correlations or no correlations between SCT symptoms and ADHD-HI symptoms have been reported (Barkley, 2014; Garner, Marceaux, Mrug, Patterson, & Hodgens, 2010; Skirbekk, Hansen, Oerbeck, & Kristensen, 2011).

SCT symptoms have been shown to be associated with specific psychosocial impairments, that is, social deficits, co-occurring psychiatric symptoms, and difficulties in neurocognitive and academic functioning after statistically controlling for ADHD-I and/or ADHD-HI symptoms (for a meta-analysis and critical review see, Becker et al., 2016a). The independent relationship between SCT symptoms and difficulties in social relationships particularly with social withdrawal beyond ADHD-I and/or ADHD-HI symptoms has been the most consistent finding (Marshall, Evans, Eiraldi, Becker, & Power, 2014; Mikami, Huang-Pollock, Pfiffner, McBurnett, & Hangai, 2007; Willcutt et al., 2014). Concerning internalizing disorders, SCT symptoms were suggested to be uniquely associated with depression (Barkley, 2013; Becker, Garner, & Byars, 2016b; Servera, Bernad, Carrillo, Collado, & Burns, 2016) and some studies have found significant associations with SCT symptoms and anxiety after statistically controlling for symptoms of ADHD-I and/or ADHD-HI (Becker, Langberg, Luebbe, Dvorsky, & Flannery, 2014; Becker, Luebbe, Fite, Stoppelbein, & Greening, 2014). Concerning externalizing disorders, in turn, SCT symptoms and ADHD-I symptoms were reported to demonstrate different correlates. A negative association between SCT symptoms and symptoms of ADHD-HI and ODD were found when symptoms of ADHD-I and anxiety/depression were statistically controlled. A positive association between symptoms of ADHD-I and anxiety/depression with ADHD-HI and ODD were reported after symptoms of SCT were statistically controlled. Regarding neurocognitive and academic functioning, slower processing speed, as well as deficits of sustained attention and impaired academic functioning were reported in individuals with SCT symptoms after statistically controlling for symptoms of ADHD-I and/or ADHD-HI (Bauermeister, Barkley, Bauermeister, Martínez, & McBurnett, 2012; Becker et al., 2016a; Lee, Burns, Snell, & McBurnett, 2014; Servera et al., 2016; Willcutt et al., 2014). Thus, research suggests that SCT symptoms are independently associated with specific psychosocial consequences above and beyond ADHD subtypes.

The majority of studies on SCT symptoms have assessed children with ADHD and TD children, and not much is known about SCT symptoms in other clinical conditions (Becker, 2014; Lee et al., 2014; Marshall et al., 2014; Servera et al., 2016; Wåhlstedt & Bohlin, 2010). Single studies on SCT symptoms have been conducted in survivors of acute lymphoblastic leukemia (Reeves et al., 2007), children with behavioral or learning disabilities (Garner et al., 2010; Garner, Mrug, Hodgens, & Patterson, 2013), psychiatrically hospitalized children (Becker, Luebbe et al., 2014; Becker, Withrow et al., 2016c), and children with sleep disorders (Becker et al., 2016b). No previous studies have been conducted on SCT in children and adolescents with HF-ASD. The lack of studies in this area is surprising taking into consideration the resemblance between symptoms and related factors of SCT and ASD. The SCT symptoms, such as “gets lost in his or her own thought”, “is slow or delayed in completing tasks”, “is underactive, slow-moving, or lacks

energy", and "seems to be in a world of his or her own" bear resemblance to behaviors that are typical in children and adolescents with ASD. In addition, the associated deficits in SCT symptoms resemble closely those found in ASD. Similarly to individuals with SCT symptoms, high rates for co-occurring internalizing psychiatric symptoms (Ghaziuddin et al., 1998; Mattila et al., 2010; Mazefsky et al., 2012; Mukaddes & Fateh, 2010), slow processing speed (Barnhill et al., 2000; Ghaziuddin & Mountain-Kimchi, 2004; Manjiviona & Prior, 1999; Planche & Lemonnier, 2012) and/or academic impairments (Estes, Rivera, Bryan, Cali, & Dawson, 2011; Griswold, 2002) have been reported in individuals with HF-ASD. If SCT symptoms are distinct from symptoms of ADHD, then an ASD+SCT group is conceivable. Based on previous studies regarding SCT symptoms in children with ADHD/other clinical groups/TD children, ASD+high levels of SCT symptoms group could be at risk for pronounced social impairments, internalizing symptoms, slower processing speed, and academic deficits in comparison to ASD group without or with low levels of SCT symptoms. Thus, recognizing SCT symptoms in ASD would be one way to plan more specific interventions for these individuals. Possibly this would serve as a preventive factor and could contribute to a better outcome.

# 2 Aims of the study

This thesis addresses neurocognitive functioning and psychiatric symptoms in children and adolescents with HF-ASD.

Studies I and II evaluated the neurocognitive performance of children and adolescents with HF-ASD in comparison to TD children and adolescents. Children and adolescents with HF-ASD were hypothesized to demonstrate difficulties in EFs and attention (the executive dysfunction account), and deficits in social perception and face recognition (the ToM deficits and the WCC accounts). These hypotheses were also based on previous studies that have reported difficulties in EFs (Happé et al., 2006; Nydén et al., 1999; Semrud-Clikeman et al., 2010), social perception (Happé, 1994; Kaland et al., 2002; Krebs et al., 2011; Rump et al., 2009), and face recognition (Klin et al., 1999; Kuusikko-Gauffin et al., 2011) in children and adolescents with HF-ASD. Additionally, children with HF-ASD (Study I) were hypothesized to show difficulties in sensorimotor skills (Ham et al., 2008; Klin et al., 1995; Manjiviona & Prior, 1995).

Study III examined psychiatric symptoms in children and adolescents with HF-ASD compared to TD children and adolescents and compared the prevalence rates of psychiatric symptoms to those reported by Ford et al. (2003). Children and adolescents with HF-ASD were hypothesized to have high rates of anxiety disorders and ADHD based on previous studies (Green et al., 2000; Lugnegård, Hallerbäck, & Gillberg, 2011; Mattila et al., 2010; van Steensel et al., 2011). Regarding the less investigated areas, children and adolescents with HF-ASD were expected to have more PTSD and eating disorder symptoms than TD children and adolescents.

Study IV assessed associations between SCT symptoms in individuals with HF-ASD to psychosocial impairments. The SCT symptoms were hypothesized to be associated with pronounced social impairments and deficits in academic functioning, higher rate of internalizing symptoms, and slower processing speed in HF-ASD based on the previous results on SCT symptoms in different developmental disorders and TD children (Bauermeister et al., 2012; Becker, Luebbe et al., 2014; Becker et al., 2016a; Becker et al., 2016b; Marshall et al., 2014; Mikami et al., 2007).

# 3 Methods

## 3.1 Participants and procedure

Participants in the HF-ASD group were children (Study I, Study III and Study IV) and adolescents with HF-ASD (Study II, Study III, and Study IV). Children and adolescents for the HF-ASD group were recruited from the Helsinki University Hospital (HUH) and from private rehabilitation and medical center focusing on neuropsychiatric disorders at Helsinki. The HUH Ethics Committee approved the study. At least one caregiver of each child and adolescent gave their written informed consent. The inclusion criteria for the HF-ASD group were: 1) Asperger syndrome diagnosis assigned clinically according to ICD-10 (World Health Organization, 1993) and 2) an age between 6 years 0 months to 16 years 1 months for Study I, Study II, and Study III; an age of 5 years 0 months to 15 years 11 months for Study IV. The clinical diagnoses of Asperger syndrome were further confirmed as a part of the research project by teams of professionals using all available information including the Autism Diagnostic Interview-Revised (Lord, Rutter, & Le Couteur, 1994; Rutter, LeCouteur, & Lord, 2003) and patient records. All children and adolescents with HF-ASD underwent cognitive capacity and neuropsychological assessments, and their parents participated in diagnostic interviews regarding ASD symptoms and symptoms of psychiatric disorders. For this study, parents also filled out the FTF questionnaire (Korkman et al., 2005) and a questionnaire regarding background information. The participants in the HF-ASD group had no genetic or major neurological disorders.

Participants in the TD groups were 1) children (Study I) and adolescents (Study II) selected from the Finnish standardization sample of the NEPSY-II (Korkman, Kirk, & Kemp, 2008) and 2) children and adolescents (Study III) recruited for study from mainstream schools belonging to the area of the Hospital District of the Helsinki and Uusimaa (HUS). Children and adolescents were selected from the standardization sample of the NEPSY-II (Korkman et al., 2008) to match the HF-ASD group with respect to gender, age, and maternal educational level. For children (Study I), two TD children per each child with HF-ASD were selected randomly of the potential TD children. For adolescents (Study II), one TD adolescent per each adolescent with HF-ASD was selected randomly of the potential TD adolescents. The difference between Study I and Study II regarding the TD group sample size is due to the small number of potential TD adolescents in the Finnish standardization sample of the NEPSY-II (Korkman et al., 2008). All children and adolescents in these TD groups attended regular school classes. They had no language or learning impairments, no neurological or psychiatric diagnoses according to parent reports. Regarding the TD group (Study III) recruited from the schools in the HUS area, at least one caregiver for each child and adolescent gave their written informed consent. The parents of this TD group were interviewed regarding psychiatric symptoms, and they filled out a questionnaire regarding background information. Children and adolescents in this TD group had no language or learning impairments, no neurological or psychiatric diagnoses according to parent reports.

Participants for the HF-ASD+High SCT, HF-ASD+Medium SCT, and HF-ASD+Low SCT subgroups (Study IV) were selected from the HF-ASD group. Two parent-rated hypoactivity items in the FTF questionnaire (Korkman et al., 2005) 1) "often in "own world" or daydreaming" and 2) "seems slow, inert, or lacking energy" were used to classify children and adolescents with HF-ASD into these three groups.

## 3.2 Neuropsychological assessment

Cognitive capacity was investigated with eight subtests of the WISC-III (Wechsler, 1991) in children and adolescents with HF-ASD. The subtests were Arithmetic, Comprehension, Information, Similarities, Block Design, Coding, Picture Completion, and Object Assembly. Previous clinical assessments of the WISC-III performance were used if these had been administered ≤ 2 years before the study.

Psychometric and clinical rationale and previous studies regarding intellectual performance in HF-ASD were used in the selection of the WISC-III subtests. Eight subtests were selected based on psychometric rationale to enable more reliable and valid estimates of IQs compared to shorter WISC-III versions (Sattler, 2001). Four subtests from verbal and performance domains were conducted to assess these domains equally. The specific WISC-III subtests were selected on the basis of clinical rationale and previous research regarding cognitive strengths and weaknesses in HF-ASD, (Barnhill et al., 2000; Ghaziuddin & Mountain-Kimchi, 2004; Noterdaeme et al., 2010) and subtests administration time.

Neurocognitive functioning was assessed with the NEPSY-II (Korkman et al., 2008). The NEPSY-II consists 29 subtests divided into six domains. Two to four subtests from each domain were administered (Table 1). The theories of executive dysfunction account, WCC, and ToM deficits account on ASD, and previous studies concerning HF-ASD were used in the selection of the NEPSY-II (Korkman et al., 2008) subtests. Additionally, the results of the "special group" study reported in the NEPSY-II United States edition (Korkman, Kirk, & Kemp, 2007) regarding children with Asperger syndrome influenced also to the selection of the NEPSY-II (Korkman et al., 2008) subtests for the present study. Previous clinical assessments of the NEPSY-II (Korkman et al., 2008) were used if these had been administered ≤ 2 years before the study.

The WISC-III and NEPSY-II subtests were administered by two clinical neuropsychologists, three psychologists, and one Master's level psychology student. The WISC-III and NEPSY-II subtests were scored by the person who administered the subtests. Training and supervision was provided for professionals who were less experienced in the administration and scoring of the WISC-III and NEPSY-II subtests. Each participant was assessed by one person.

**Table 1** *NEPSY-II[1] subtests used in Study I and Study II.*

| NEPSY-II[1] subdomains and subtests | Study I | Study II |
|---|---|---|
| Attention and EF | | |
| Auditory Attention and Response Set combined | - | x |
| Auditory Attention | x | x |
| Response Set | x | x |
| Design Fluency | x | x |
| Inhibition and Switching combined | - | x |
| Inhibition, Naming | x | - |
| Inhibition, Inhibition | x | - |
| Inhibition, Switching | x | - |
| Visual Attention | x | x |
| Language | | |
| Comprehension of Instructions | x | x |
| Word Generation | x | x |
| Memory and Learning | | |
| Memory for Designs combined | x | x |
| Memory for Faces combined | x | x |
| Narrative Memory | x | x |
| Sensorimotor | | |
| Imitating Hand Positions | x | x |
| Visuomotor Precision | x | x |
| Social Perception | | |
| Affect Recognition | x | x |
| Theory of Mind combined | x | x |
| Visuospatial | | |
| Design Copying | x | x |
| Geometric Puzzles | x | x |
| Picture Puzzles | x | x |

[1]NEPSY-II = a developmental neuropsychological assessment (Korkman et al., 2008).

## 3.3 Psychiatric symptom assessment

Psychiatric symptoms were assessed with the online version (see www.dawba.com) of the Development and Well-Being Assessment (DAWBA) (Goodman, Ford, & Richards, 2000) in the HF-ASD group and in the TD group. The DAWBA is a structured interview covering comprehensively questions of common psychiatric disorders based on DSM-IV (American Psychiatric Association, 1994) and ICD-10 (World Health Organization, 1993) diagnostic criteria (Goodman et al., 2000). After screening questions, fixed response option-based questions are displayed, and semi-structured information is gathered with open-ended questions to obtain the participants' descriptions of the difficulties.

The Finnish version of the DAWBA conducted in the present study consisted of the following DAWBA subdomains: Background; Development of language, routines, play, and social ability; Depression; Dieting, bingeing and concern about body shape; Difficult or troublesome behavior; Hyperactivity and attention problems; Irritability and temper and anger control; More about strengths and good points; Obsessions and compulsions; Other concerns; Panic attacks; Specific fears; Social fears; Stress after a very frightening event; Tics; Worries about separation from key "attachment figures"; and Worrying a lot about many different things. In this study, information was collected from parents. One parent or

both parents were interviewed depending on who was/were eligible to participate in study. The DAWBA interviews were conducted by one psychologist, one Master's level psychology student, and one nurse. The diagnoses were assigned by an experienced child psychiatrist according DSM-IV (American Psychiatric Association, 1994) and ICD-10 (World Health Organization, 1993) criteria based on the DAWBA results. The DSM-IV diagnoses are reported to assist comparison to other studies. In Study III all of the aforementioned subdomains were utilized. In Study IV internalizing symptoms were assessed with DAWBA subdomains of Depression; Obsessions and compulsions; Panic attacks; Specific fears; Social fears; Stress after a very frightening event; Worries about separation from key "attachment figures", and Worrying a lot of about many different things. Externalizing symptoms were evaluated with DAWBA subdomains of Irritability and temper and anger control and Difficult and troublesome behavior in Study IV.

## 3.4 Developmental and behavioral assessment

SCT symptoms, academic functioning, and social skills in the HF-ASD group in Study IV were examined with the FTF questionnaire filled out by parents (Kadesjö et al., 2004; Korkman et al., 2005; Korkman, Jaakkola, Ahlroth, Pesonen, & Turunen, 2004). The FTF consists of 181 questions which are scored on a three-point Likert scale: 0 = "does not apply"; 1 = "applies sometimes or to some extent"; and 2 = "definitely applies" (Kadesjö et al., 2004). Higher scores in the FTF signify more impairments. Eight developmental and behavioral domains are covered in the FTF: 1) Emotional/behavioral problems, 2) Executive functions, 3) Language, 4) Learning, 5) Motor skills, 6) Memory, 7) Perception, and 8) Social skills. The domains are further divided into subdomains (Kadesjö et al., 2004; Korkman et al., 2005; Korkman et al., 2004).

SCT symptoms were assessed with two items of the Hypoactivity subdomain of the Executive function domain of the FTF: 1) "often in "own world" or daydreaming", and 2) "seems slow, inert, or lacking energy". These two FTF items bear a high resemblance to the relevant SCT rating scale items (Penny, Waschbusch, Klein, Corkum, & Eskes, 2009). The first FTF item "often in "own world" or daydreaming" resembles the SCT items "seems to be in a world of his or her own" and "daydreams" closely. The second FTF item "seems slow, inert, or lacking energy" resembles the SCT item "is underactive, slow-moving, or lacks energy" closely. These SCT rating scale items load highly on two of the three SCT factors ("Slow", "Daydreamer", "Sleepy"), that is, on the "Daydreamer" (.823 and .872) and on the "Sleepy" (.825) SCT factors (Penny et al., 2009). Children and adolescents with HF-ASD were divided into three groups based on these two items: 1) the ASD+High SCT group (sum of 3 or 4 on the two Hypoactivity items), 2) the ASD+Medium SCT group (sum of 2 on the two Hypoactivity items), and 3) to the ASD+Low SCT group (sum of 0 or 1 on the two Hypoactivity items).

Questions covering the Learning and Social skills domains were used to examine academic functioning and social skills. With respect to the Learning subdomain, only participants aged 9 to 15 years were selected for the analyses because the FTF contains norms only for this age range on the Learning subdomain. Finally, a sum of raw scores on

the Attention and concentration subdomain and the Overactivity and impulsivity subdomain of the FTF were utilized for controlling symptoms of ADHD in the analyses.

## 3.5 Statistical analyses

Chi-square tests, Fisher's exact tests, independent samples *t*-tests, and Mann Whitney *U* – tests were used to compare the background variables of the groups. The statistical significange level in Studies I-IV was set at $p < .05$. Main variables of Studies I-IV can be found in Table 2.

**Table 2** *Main assessment methods and variables of the Studies I-IV.*

| Main variables of the Studies I-IV | Study I | Study II | Study III | Study IV |
|---|---|---|---|---|
| ADI-R[1] confirmation of clinical diagnose of AS | x | x | x | x |
| WISC-III[2] subtests Background variable (FSIQ) | x | x | x | x |
| Dependent variable (VIQ and PIQ) | x | x | - | - |
| Dependent variables (8 subtests) | - | x | - | - |
| Dependent variable (only the Coding subtest) | - | - | - | x |
| NEPSY-II[3] Dependent variables (16 subtests) | x | x | - | - |
| DAWBA[4] | | | | |
| Background variable (Attention and hyperactivity subdomain) | - | - | - | x |
| Dependent variables (17 psychiatric symptom subdomains) | - | - | x | - |
| Independent variables | - | - | - | x |
| (11 psychiatric symptom subdomains in two categories) | | | | |
| FTF[5] | | | | |
| Independent and dependent variable[6] | - | - | - | x |
| (SCT items from the Hypoactivity subdomain; | | | | |
| HF-ASD+High SCT, HF-ASD+Medium SCT, HF-ASD+Low SCT) | | | | |
| Covariate | - | - | - | x |
| (symptoms from the Attention and Hyperactive-Impulsive subdomain) | | | | |
| Dependent variable (Learning domain) | - | - | - | x |
| Dependent variable (Social skills domain) | - | - | - | x |

AS = Asperger syndrome; FSIQ = full scale intelligence quotient; VIQ = verbal intelligence quotient; PIQ = performance intelligence quotient; SCT = sluggish cognitive tempo.

[1]ADI-R = autism diagnostic interview-revised (Lord et al., 1994; Rutter et al., 2003).

[2]WISC-III = Wechsler Intelligence Scales, third edition (Wechsler, 1991).

[3]NEPSY-II = a developmental neuropsychological assessment (Korkman et al., 2008).

[4]DAWBA = development and well-being assessment (Goodman et al., 2000).

[5]FTF = five-to fifteen questionnaire (Korkman et al., 2005).

[6] The group was either independent or dependet variable determined by the analysis used.

With respect to cognitive capacity, one-sample *t*-tests were conducted to compare the WISC-III scores of children and adolescents with HF-ASD to the population mean (*M* =

100) (Studies I-II). Paired samples *t*-tests were applied to investigate the difference of VIQ-PIQ in children and adolescents with HF-ASD (Studies I-II). Concerning neuropsychological functioning (Studies I-II), a multivariate analysis of variance (MANOVA) was used to compare the groups and follow-up analyses for each NEPSY-II (Korkman et al., 2008) subtest were conducted using *t*-tests. Multiple comparisons were not corrected because this procedure increases the probability of Type II error (i.e., not rejecting a false $H_0$) and reduces power (Nakagawa, 2004; Perneger, 1998). Effect sizes were calculated to assist the interpretation of the results. For MANOVA, effect sizes were computed using the partial eta squared and for one-samples/paired-samples/independent samples *t*-tests effect sizes were computed using the Cohen's *d*.

Group differences in psychiatric symptoms (Study III) were analysed with chi-square tests and Fisher's exact tests. Differences between the percentage of co-occurring psychiatric symptoms in individuals with HF-ASD and the reported prevalence rates by Ford et al. (2003) were analysed with one-sample binomial tests (Study III).

Regarding SCT (Study IV), symptoms of ADHD-I and ADHD-HI were statistically controlled in all analyses and when appropriate age was also included as a covariate. Group differences (ASD+High SCT, ASD+Medium SCT and ASD+Low SCT) in social skills, academic functioning, and processing speed were studied with three separate univariate analyses of covariance (ANCOVAs). Two logistic regression analyses were conducted to assess group differences (ASD+High SCT, ASD+Medium SCT and ASD+Low SCT) in internalizing and externalizing psychiatric symptoms.

# 4 Results

## 4.1 Participant characteristics

The participant characteristics in Studies I-IV can be found in Table 3. In Studies I-III, children and adolescents with HF-ASD did not differ statistically significantly from TD children and adolescents in age, gender, or parental education level.

In Study IV, the sample was divided into three groups on the basis of SCT symptoms: the HF-ASD+High SCT group ($n$ = 17), the HF-ASD+Medium SCT group ($n$ = 18), and the HF-ASD+Low SCT group ($n$ = 20). These groups differed statistically significantly in age [$\chi^2(2, N = 55) = 6.6$, $p = .036$], ADHD or Hyperactivity evaluated with the DAWBA (Fisher's exact test, $p = .04$), and symptoms in ADHD-I [$F(2, 52) = 4.3$, $p = .018$] evaluated with the FTF. Participants in the ASD+Medium SCT group were older compared to the participants in the ASD+Low SCT group [$\chi^2$ (1, $N$ = 38) = 5.9, $p$ = .015]. The ASD+High SCT group had higher levels of ADHD-I symptoms compared to the ASD+Medium SCT [$t(52) = -2.5$, $p = .015$], and ASD+Low SCT groups [$t(52) = -2.6$, $p$ = .011]. No statistically significant group differences were found in gender, parent education level, full scale intelligence quotient (FSIQ), or symptoms of ADHD-HI evaluated with the FTF. Regarding analyses conducted with smaller sample size on the Learning domain of the FTF, 13 participants were classified in the ASD+High SCT group, 18 participants were classified in the ASD+Medium SCT group, and 12 participants were classified in the ASD+Low SCT group. The groups differed statistically significantly in symptoms of ADHD-I evaluated with the FTF [$F(2, 40) = 4.8$, $p = .014$]. The ASD+High SCT group had statistically higher level of symptoms of ADHD-I in comparison to the ASD+Medium SCT group [$t(40) = -2.2$, $p = .032$] , and ASD+Low SCT groups [$t(40) = -3.0$, $p = .005$]. No statistically significant differences were found in age, gender, parent education level, FSIQ, and symptoms of ADHD-HI evaluated with the FTF.

## 4.2 Neurocognitive functioning

Children with HF-ASD (Study I) had a significantly higher FSIQ than the population mean [$t(29) = 2.26$, $p = .031$, $d = 0.48$]. No significant differences in FSIQ in adolescents with HF-ASD were found in comparison to the population mean (Study II).

Both children [$t(29) = 3.07$, $p = .005$, $d = 0.75$] and adolescents with HF-ASD [$t(29) = 4.40$, $p < .001$, $d = 0.80$] had a significantly higher VIQ compared to the population mean while no significant differences between PIQ and the population mean were found in these individuals (Studies I-II). A trend for a higher VIQ compared to PIQ was found in children with HF-ASD ($p = .05$) (Study I). Adolescents with HF-ASD had a significantly higher VIQ in comparison to PIQ ($p < .001$) (Study II).

**Table 3** *Participant characteristics in Studies I-IV.*

| | Study I | | Study II | | Study III | | Study IV | | |
|---|---|---|---|---|---|---|---|---|---|
| | HF-ASD ($n$ = 30) | TD ($n$ = 60) | HF-ASD ($n$ = 30) | TD ($n$ = 30) | HF-ASD ($n$ = 60) | TD ($n$ = 60) | HF-ASD+ High SCT ($n$ = 17) | HF-ASD+ Medium SCT ($n$ = 18) | HF-ASD+ Low SCT ($n$ = 20) |
| FSIQ $M$ (SD) | 107.2 (17.3) | - | 103.2 (10.7) | - | 105.5 (14.5) | - | 100.4 (11.7) | 104.2 (12.1) | 109.3 (17.7) |
| Gender (boys:girls) | 28:2 | 56:4 | 20:10 | 20:10 | 48:12 | 47:13 | 14:3 | 12:6 | 17:3 |
| Age years $M$ (SD) | 9.1 (1.3) | 9.1 (1.4) | 13.5 (1.2) | 13.7 (1.0) | 11.6 (2.5) | 11.1 (2.8) | 11.1 (2.7) | 12.9 (1.7) | 10.6 (2.8) |
| Maternal education $n$ (%) | | | | | | | | | |
| Lower | 2 (6.7) | 4 (6.7) | 3 (10.0) | 2 (6.7) | 4 (6.7) | 2 (3.3) | 0 (0.0) | 3 (16.7) | 1 (5.0) |
| Medium | 9 (30.0) | 18 (30.0) | 16 (53.3) | 17 (56.7) | 26 (43.3) | 18 (30.0) | 10 (58.8) | 5 (27.8) | 8 (40.0) |
| Higher | 19 (63.3) | 38 (63.3) | 11 (36.7) | 11 (36.7) | 30 (50.0) | 40 (66.7) | 7 (41.2) | 10 (55.6) | 11 (55.0) |

HF-ASD = higher functioning autism spectrum disorder, TD = typically developing, High SCT = high levels of sluggish cognitive tempo symptoms, Medium SCT = medium levels of sluggish cognitive tempo symptoms, Low SCT = low levels of sluggish cognitive tempo symptoms, FSIQ = full scale intelligence quotient.

Note. Maternal education: Lower = primary school with or without general secondary school or comprehensive school, Medium = upper secondary education, comprising vocational and general education, Higher = polytechnics or university education.

Children [Wilk's lambda = 0.53, $F(19, 70) = 3.28$, $p < .001$, $\eta_p^2 = 0.47$] and adolescents with HF-ASD [Wilk's lambda = 0.48, $F(16, 43) = 2.96$, $p = .002$, $\eta_p^2 = 0.52$] (Studies I-II) differed in neuropsychological measures from TD children and adolescents (Figure 1, Figure 2). Follow-up analyses showed that in children with HF-ASD six NEPSY-II subtests and in adolescents with HF-ASD four NEPSY-II subtests differentiated the groups.

Both children and adolescents with HF-ASD (Studies I-II) had significantly lower scores in the Response Set subtest [$t(82) = 3.74$, $p < .001$, $d = 0.87$; $t(58) = 4.11$, $p < .001$, $d = 1.06$, respectively], Memory for Faces subtest [$t(88) = 2.24$, $p = .028$, $d = 0.50$; $t(58) = 2.80$, $p = .007$, $d = 0.72$, respectively], and Design Copying subtest [$t(88) = 2.28$, $p = .025$, $d = 0.51$; $t(58) = 2.67$, $p = .010$, $d = 0.69$, respectively] compared to the TD groups. Children with HF-ASD had a trend towards lower performance in the Visuomotor Precision subtest compared to TD children [$t(36.9) = 2.01$, $p = .051$, $d = 0.55$] while adolescents with HF-ASD had a significantly lower score in the Visuomotor Precision subtest in comparison to TD adolescents [$t(58) = 3.28$, $p = .002$, $d = 0.85$]. Additionally, children with HF-ASD were found to have lower scores in the Word Generation [$t(88) = 2.68$, $p = .009$, $d = 0.60$], Narrative Memory [$t(72) = 2.69$, $p = .009$, $d = 0.64$], and Imitating Hand Positions [$t(88) = 2.62$, $p = .01$, $d = 0.59$] subtests compared to the TD group.

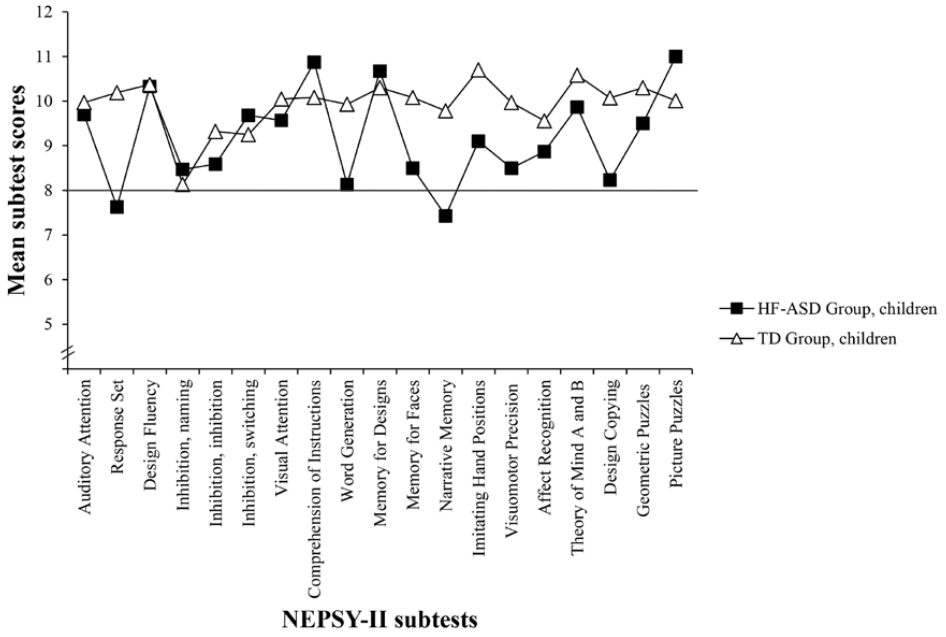


**Figure 1** *Neurocognitive functioning in children with HF-ASD and TD children in the NEPSY-II subtests (Korkman et al., 2008). Standard scores ≥ 8 signify average or above average neurocognitive functioning.*

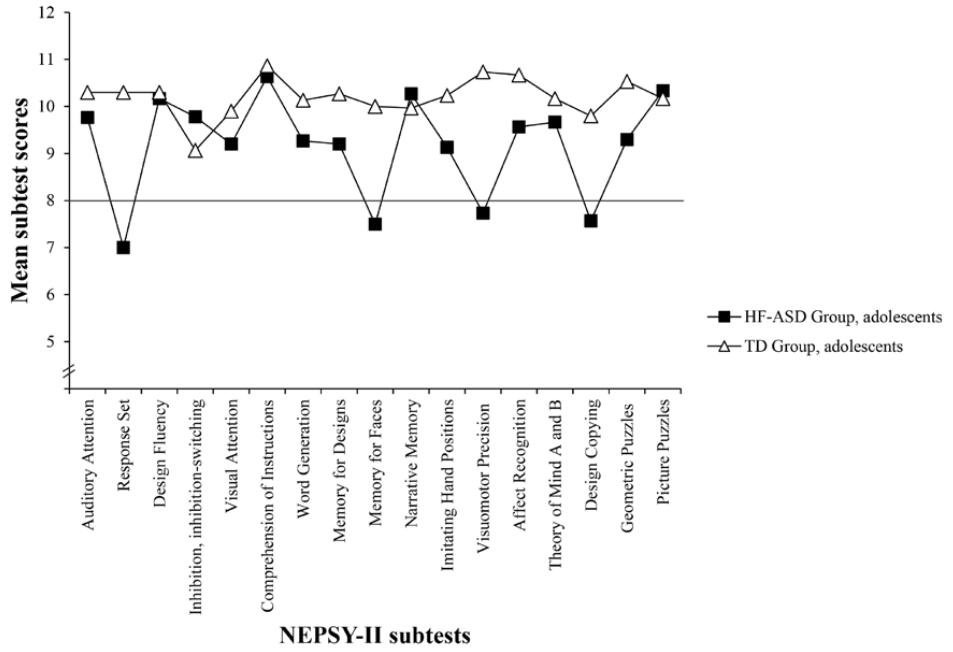


**Figure 2** *Neurocognitive functioning in adolescents with HF-ASD and TD adolescents in the NEPSY-II subtests (Korkman et al., 2008). Standard scores ≥ 8 signify average or above average neurocognitive functioning.*

## 4.3 Psychiatric symptoms

As seen from Table 4, children and adolescents with HF-ASD had significantly higher rate of psychiatric symptoms in comparison to the TD group and in comparison to the prevalence rate found by Ford et al. (2003). Children and adolescents with HF-ASD had a significantly higher percentage of emotional disorders, ADHD/hyperkinesis, and tic disorders in comparison to TD children and adolescents. Follow-up analyses showed a significantly higher rate of specific phobia and other tic disorders in the HF-ASD group compared to the TD group. In comparison to the Ford et al. (2003) study, the HF-ASD group had a significantly higher percentage of specific phobia, generalized anxiety disorder (GAD), OCD, and other depression. Additionally, the HF-ASD group had a significantly higher rate of ADHD-C, ADHD-I, and ADHD-HI in comparison to the prevalence rates reported by Ford et al. (2003). Furthermore, the HF-ASD group had a significantly higher percentage of ODD and tic disorders compared to the prevalence rates of Ford et al. (2003).

Additionally, it was assessed whether single or multiple psychiatric disorder symptoms were present in the HF-ASD group and in the TD group. This was done by calculating the percentage of participants endorsing one, two, three, or four psychiatric disorders within each group (HF-ASD or TD). In the HF-ASD group, 40% had one psychiatric disorder;

20% had two psychiatric disorders; 7% had three psychiatric disorders; and 2% had four psychiatric disorders. In the TD group, 10% had one psychiatric disorder; 2% had two psychiatric disorders; and 0% had three or four psychiatric disorders. In comparison, in the Ford et al. (2003) study, 7% had one psychiatric disorder; 2% had two psychiatric disorders; 0.5% had three psychiatric disorders; and 0.2% had four psychiatric disorders.

**Table 4** *Percentage of co-occurring psychiatric symptoms in children and adolescents with HF-ASD and TD children and adolescents in the DAWBA*[1].

| Psychiatric disorder | ASD % | TD % | Ford et al. (2003) | $p^2$ | $p^3$ |
|---|---|---|---|---|---|
| Any disorder | 68.3 | 11.7 | 9.5 | < .001 | < .001 |
| Any emotional disorder | 36.7 | 5.0 | – | < .001 | – |
| Specific phobia | 20.0 | 0.0 | 1.0 | < .001 | < .001 |
| Generalized anxiety disorder | 10.0 | 1.7 | 0.7 | *ns* | < .001 |
| Obsessive-compulsive disoder | 8.3 | 0.0 | 0.3 | *ns* | < .001 |
| Post-traumatic stress disorder | 1.7 | 0.0 | 0.1 | *ns* | *ns* |
| Separation anxiety | 1.7 | 0.0 | 1.2 | *ns* | *ns* |
| Social phobia | 1.7 | 0.0 | 0.3 | *ns* | *ns* |
| Panic disorder | 0.0 | 0.0 | 0.1 | – | *ns* |
| Agoraphobia | 0.0 | 0.0 | 0.1 | – | *ns* |
| Other anxiety disorder | 0.0 | 0.0 | 0.9 | – | *ns* |
| Other depression | 5.0 | 1.7 | 0.2 | *ns* | < .001 |
| Major depression | 1.7 | 0.0 | 0.7 | *ns* | *ns* |
| Undifferentiated anxiety/depression | 0.0 | 1.7 | – | *ns* | – |
| Any ADHD/hyperkinesis | 26.7 | 1.7 | – | < .001 | – |
| ADHD combined | 11.7 | 1.7 | 1.4 | *ns* | < .001 |
| ADHD inattentive | 8.3 | 0.0 | 0.7 | *ns* | < .001 |
| ADHD hyperactive-impulsive | 6.7 | 0.0 | 0.2 | *ns* | < .001 |
| Other hyperactivity | 0.0 | 0.0 | – | – | – |
| Any behavioral disorder | 10.0 | 3.3 | – | *ns* | – |
| Oppositional defiant | 8.3 | 3.3 | 2.3 | *ns* | < .01 |
| Conduct disorder | 1.7 | 0.0 | 1.5 | *ns* | *ns* |
| Other disruptive | 0.0 | 0.0 | 1.1 | – | *ns* |
| Any tic disorder | 18.3 | 1.7 | 0.1 | < .01 | < .001 |
| Tourette | 5.0 | 1.7 | – | *ns* | – |
| Chronic | 3.3 | 0.0 | – | *ns* | – |
| Other | 10.0 | 0.0 | – | < .05 | – |
| Any eating disorder (anorexia, bulimia, other) | 0.0 | 0.0 | 0.1 | – | *ns* |
| Any other disorder | 1.7 | 1.7 | – | *ns* | – |

[1] DAWBA = Development and Well-Being Assessment (Goodman et al., 2000).

[2] HF-ASD group in comparison to the TD group.

[3] HF-ASD group in comparison to the Ford et al. (2003) prevalence rates.

On a more detailed level, the three most endorsed specific fears in the ASD+specific fear ($n$ = 12) group were fear of blood, infection, or injury (66.7%), fear of animals (58.3%), and fear of the dark (58.3%). The three most common GAD symptoms in the ASD+GAD group ($n$ = 6) were worries regarding school work or examinations and making and keeping friends. Regarding depression, symptom of being sad was endorsed in all individuals (100%) in the ASD+depression group ($n$ = 4; major depression and other depression). Irritability and losing interest was present (25%) in only one participant in the ASD+depression group. Repetitive actions (50%) and ordering and symmetry (50%)

occurred most often in the ASD+OCD group ($n$ = 5). The most common ODD symptoms in the ASD+ODD group ($n$ = 15) were arguing with adults (80%) and blaming others for one's own acts (80%). As regards to tic symptoms in the ASD+tic disorder group ($n$ = 11), throat clearing (54.5%), and making little noises (54.5%) occurred most often. Finally, one participant in the HF-ASD group had a limited food repertoire.

## 4.4 Sluggish cognitive tempo

On the FTF Social skills domain, a significant effect of group was found [$F(2,49) = 7.2$, $p = .002$, $\eta_p^2 = 0.23$] after statistically controlling for symptoms of ADHD-I and ADHD-HI and age. More detailed analyses showed that the ASD+High SCT [$t(49) = -3.5$, $p$=.001] and ASD+Medium SCT groups [$t(49) = -3.0$, $p = .004$] had significantly higher scores (i.e., more difficulties) on the FTF Social skills domain compared to the ASD+Low SCT group. On the FTF Learning domain, no significant group effect was found when controlling for symptoms of ADHD-I and ADHD-HI and age.

With respect to internalizing symptoms evaluated with the DAWBA, the group predicted marginally significantly internalizing symptoms [Wald $\chi^2(2) = 5.1$, $p = .08$] when ADHD-I and ADHD-HI symptoms and age were statistically controlled. The ASD+High SCT was significantly [$b = 1.9$, Wald $\chi^2(1) = 4.8$, $p = .028$] and the ASD+Medium SCT group was marginally significantly [$b = 1.4$, Wald $\chi^2(1) = 2.8$, $p = .094$] more likely to have symptoms of internalizing disorders compared to the ASD+Low SCT group. No significant associations between group and externalizing symptoms assessed with the DAWBA were found when symptoms of ADHD-I and ADHD-HI and age were statistically controlled. Finally, the effect of group on processing speed evaluated with the Coding subtest of the WISC-III was non-significant after statistically controlling for symptoms of ADHD-I and ADHD-HI.

# 5 Discussion

The four studies of this thesis aimed at assessing neurocognitive functioning, psychiatric symptoms, and SCT in children and adolescents with HF-ASD. The main findings were as follows. First, the neurocognitive impairments in children and adolescents with HF-ASD were mild when assessed with clinical neuropsychological subtests. This was evident at group level with the mean standard scores in the HF-ASD group corresponding mainly borderline performance (i.e. mild deficits) in the neurocognitive areas in which deficits were found compared to TD participants. Second, high rates of psychiatric disorders in individuals with HF-ASD were found. Third, SCT in individuals with ASD was associated with more pronounced social impairments and higher rates of internalizing symptoms. The following sections focus on detailed discussion of Studies I-IV.

## 5.1 Neurocognitive functioning in HF-ASD

The assessments on cognitive capacity showed that children and adolescents with HF-ASD had strengths in VIQ in comparison to the population means in the WISC-III (Wechsler et al., 1991). The VIQ-PIQ discrepancy approached statistical significance in children with HF-ASD, and in adolescents with HF-ASD the VIQ-PIQ discrepancy was significant. These results are in line with previous studies assessing cognitive capacity in children and adolescents with HF-ASD (Cederlund, 2004; Ghaziuddin & Mountain-Kimchi, 2004; Ozonoff et al., 2000). Regarding VIQ, the highest scores were found in the Information and Similarities subtests both in children and adolescents with HF-ASD which is consistent with previous studies conducted in children and adolescents with Asperger syndrome (Barnhill et al., 2000; Ghaziuddin & Mountain-Kimchi, 2004; Manjiviona & Prior, 1999; Noterdaeme et al., 2010; Planche & Lemonnier, 2012).

Neurocognitive profiles of children and adolescents with HF-ASD differed significantly from those of TD children and TD adolescents, respectively (Studies I-II). Follow-up analyses showed neurocognitive impairments in three areas in children and adolescents with HF-ASD, these being attention and EFs, face recognition and visuomotor functions. The first and most pronounced neurocognitive deficit was found in the domain of attention and EF in set-shifting both in children and adolescents with HF-ASD. This was demonstrated by lower performance in the Response Set subtest of the NEPSY-II in children and adolescents with HF-ASD in comparison to TD groups with large effect sizes while no significant group differences were found in the Auditory Attention subtest of the NEPSY-II. The Auditory Attention subtest (Part A) is designed to assess selective and sustained attention and the Response Set subtest (Part B) is designed to assess selective and sustained attention and set-shifting. Thus, the results of Study I and Study II indicate that children and adolescents with HF-ASD had challenges specifically in set-shifting in comparison to TD children and TD adolescents. Fifty-three percent of children and adolescents with HF-ASD had below average scores (age-corrected scaled score < 8) in the Response Set subtest.

These results are generally in line with previous studies reporting set-shifting deficits in individuals with HF-ASD (Corbett, Constantine, Hendren, Rocke, & Ozonoff, 2009; Ozonoff et al., 2000; Semrud-Clikeman, Walkowiak, Wilkinson, & Butcher, 2010). No significant group differences were found with respect to commission or omission errors in the Response Set subtest of the NEPSY-II when children and adolescents with HF-ASD were compared to TD children and adolescents respectively. Thus, lower performance in the Response Set subtest of the NEPSY-II in children and adolescents with HF-ASD was explained by their slower reactions to the target stimuli. This is line with previous studies that have reported that children and adolescents with HF-ASD have deficits in information processing when the amount of information is increased (Minshew et al., 1997; Williams, Goldstein, & Minshew, 2005; Yoran-Hegesh, Kertzman, Vishne, Weizman, & Kotler, 2009). Additionally, auditory processing deficits can partly explain the results. Lepistö et al. (2006) reported a speech-specific impairment in involuntary auditory orienting in children with Asperger syndrome. This deficit was present even in adults with Asperger syndrome (Lepistö, Nieminen-von Wendt, von Wendt, Näätänen, & Kujala, 2007).

Only children with HF-ASD (Study I) scored lower than TD children in the Word generation subtest and Narrative memory subtest of NEPSY-II while in adolescents with HF-ASD group differences were not found (Study II). Although in NEPSY-II the Word generation subtest belongs to the Language subdomain and the Narrative memory subtest belongs to the Memory subdomain, both of these subtest types have also attention and EF component (Ropacki & Perry, 2007; Shing et al., 2010; Stevens & Grady et al., 2007). Therefore, children and adolescents with primary difficulties in attention and EFs could perform poorly in these subtests although their language and memory functions are intact. Since children with HF-ASD had strengths in verbal reasoning in the present thesis, it is more likely that difficulties in attention and EFs, and slower processing speed are related to their lower performance in these subtests. These results are consistent with previous studies reporting difficulties in verbal fluency and narrative memory in individuals with HF-ASD (Diehl et al., 2006; Losh & Gordon, 2014; Verté, Geurts, Roeyers, Oosterlaan, & Sergeant, 2006).

In the attention and EF domain no other significant group differences were found in children and adolescents with HF-ASD. Overall, the research results have been mixed in previous studies assessing EFs in ASD and some studies have reported intact EFs in these individuals (for a meta-analysis, see Demetriou et al., 2017; for a review, see Kenworthy, Yerys, Anthony, & Wallace, 2008). This is surprising since the majority of clinicians, parents, and teachers agree that individuals with ASD have EF difficulties in their daily living (Kenworthy et al., 2008). In a recent meta-analysis of EFs in ASD, Demetriou et al. (2017) found evidence supporting a broad EF deficit in ASD that is relatively stable throughout the development. In Demetriou et al. (2017) meta-analysis the neuropsychological assessment methods of EFs, however, had a limited utility in differentiating individuals with ASD from TD participants and mainly a questionnaire-based assessment method, the Behavioral Rating Inventory of Executive Function (BRIEF) (Gioia, Isquith, & Kenworthy, 2000) was able to reach clinical sensitivity. Indeed, questionnaire-based assessment methods are relevant with respect to ecological validity (i.e. how the results apply to real-world situations). It is noteworthy, however, that

questionnaires can have even greater interpretational difficulties compared to EF tasks (Snyder, Miyake, & Hankin, 2015). In future studies, the use of multiple tasks to assess the specific EF components and the use of sensitive cognitive neuroscience and cognitive psychology EF tasks alongside clinical neuropsychological tests would be helpful to alleviate the EF task impurity problem (i.e. EF tasks always involve also non-EF processes) as has been recommended by Snyder, Miyake and Hankin (2015).

The second neurocognitive deficit in children and adolescents with HF-ASD was face recognition memory. Children and adolescents with HF-ASD scored significantly lower than TD children and adolescents in the Memory for Faces subtest of the NEPSY-II. Thirty-three percent of children with HF-ASD and 53% of adolescents with HF-ASD scored below average on this subtest. These findings are consistent with studies that have assessed facial memory in children and adults with HF-ASD (Klin et al., 1999; Kuusikko-Gauffin et al., 2011; O'Hearn et al., 2010). However, the results of face recognition memory in adolescents with HF-ASD in Study II contradict those of Kuusikko-Gauffin et al. (2011) study partly. They assessed facial recognition memory with the Memory for Faces subtest of the NEPSY (Korkman, Kirk, & Kemp, 1997) which is the previous version of the Memory for Faces subtest of the NEPSY-II (Korkman et al., 2008). Kuusikko-Gauffin et al. (2011) found that younger individuals with HF-ASD scored lower in the Memory for Faces subtest while no significant group differences were found regarding older children with HF-ASD. In Study I and Study II, a newer version of the Memory for Faces subtest from the NEPSY-II (Korkman et al., 2008) was used that has been developed to be a more sensitive measure of facial recognition memory in comparison to the Memory for Faces subtest of the NEPSY (Korkman et al., 1997). Photographs have been modified to show only faces, and a number of characteristics (e.g., background shading, hair) that could help to recognize faces have been removed or decreased from the Memory for Faces subtest of the NEPSY-II (Korkman et al., 2007; Korkman et al., 2008). This could explain the mixed results.

Contrary to expectations, no significant group differences were found in the domain of Social perception, that is, in the Affect Recognition or Theory of mind subtests of the NEPSY-II in children and adolescents with HF-ASD in comparison to the TD groups (Studies I-II). Overall, studies regarding emotional expression recognition in individuals with HF-ASD have been mixed (Krebs et al., 2011; Lindner & Rosen, 2006; Tracy et al., 2011). One possibility for the non-significant results in Studies I-II is that the Affect Recognition subtest was not sensitive enough to detect deficits in recognition of emotion expressions in children and adolescents with HF-ASD. The response time is unlimited in the Affect Recognition subtest of the NEPSY-II. There is an indication that emotion recognition deficits are observed in individuals with HF-ASD only when task demands, such as processing speed, are increased (Harms, Martin, & Wallace, 2010). Children and adolescents with HF-ASD did not differ significantly from TD children and adolescents in the ToM subtests in Studies I-II. Previous studies have reported mixed results regarding the performance on ToM tasks in individuals with HF-ASD (Kaland et al., 2002; Kaland et al., 2008; Scheeren, de Rosnay, Koot, & Begeer, 2013). While some studies indicate that individuals with HF-ASD have impairments in advanced ToM tasks (Barnhill et al., 2000; Happé, 1994; Kaland et al., 2002; Kaland, Smith, & Mortensen, 2008), a more

recent large-scale study by Scheeren et al. (2013) did not find significant group differences between shool-aged children and adolescents with HF-ASD ($n$ = 194; age range 6−20 years) compared to TD children and adolescents ($n$ = 60, age range 6−17 years) in advanced theory of mind tasks. Additionally, research suggests that individuals with HF-ASD are able to pass explicit ToM tasks possibly through compensatory learning while being impaired in spontaneous mental-state attributions (Senju, 2012). The results of Studies I-II are in line with the findings with Scheeren et al. (2013). These results indicate that more sensitive and implicit measures of ToM are needed to detect ToM deficits in individuals with HF-ASD.

The third neurocognitive deficit was found in visuomotor functions in children and adolescents with HF-ASD. This was indicated by a significantly lower performance of the HF-ASD group in comparison to TD group in the Imitating Hand Positions (Study I, children with HF-ASD), Visuomotor Precision (Studies I-II, children and adolescents with HF-ASD), and Design Copying (Studies I-II, children and adolescents with HF-ASD) subtests of the NEPSY-II. These results are in line with previous studies reporting fine motor and visuomotor impairments in children and adolescents with HF-ASD (Ham et al., 2008; Hooper, Poon, Marcus, & Fine, 2006; Narzisi, Muratori, Calderoni, Fabbro, & Urgesi, 2013). Examining individual scores in the HF-ASD groups revealed that in the Imitating Hands Positions subtest 27% of children had scores below average, in the Design Copying subtest 47% of children and 43% of adolescents scored below average, and in the Visuomotor Precision subtest 50% of children and 43% of adolescents had scores below average.

In summary, the neurocognitive profiles of children and adolescents with HF-ASD differed from those of TD children and adolescents. Overall, the neurocognitive strengths and weaknesses were similar in children and adolescents with HF-ASD compared to TD children and adolescents. Uneven cognitive reasoning profile by strengths in VIQ as compared to PIQ was characteristic to children and adolescents with HF-ASD. Difficulties were found in the domain of attention and EFs, face recognition memory, and visuomotor functions. Investigation of individual scores revealed wide heterogeneity regarding neurocognitive functioning in specific NEPSY-II subtests within the HF-ASD groups.

At the theoretical level, the results of Studies I-II supported mostly the EF account of ASD, and to some extent the WCC account but not ToM deficits account. More specifically, the EF account was mainly supported by the findings of impaired set-shifting in children and adolescents with HF-ASD, and by the findings that children with HF-ASD had difficulties in verbal fluency and narrative memory. The WCC account was supported by the findings of difficulties in face recognition memory in children and adolescents with HF-ASD. The ToM deficits account was not supported due to the non-significant results of ToM performance although it should be acknowledged that the non-significant results may have resulted from limited sensitivity of the ToM subtests to detect these difficulties in HF-ASD. Overall, it is important to note, however, that neurocognitive functioning was largely intact in the present studies in children and adolescents with HF-ASD and the reported deficits in these individuals at the test-level were mild. Furthermore, high heterogeneity regarding these deficits was found with some individuals having weaknesses and some individuals performing adequately. Participants in this thesis were

rigorously diagnosed both in clinical work, as well as in this research project by multidisciplinary teams. Thus, they showed definite ASD symptoms. Based on the neurocognitive accounts of ASD one would expect participants with clear ASD symptoms to show clear deficits in EFs, ToM, and WCC related tasks. This was not the case in Studies I-II. These results indicate that the impairments in EFs, ToM, and CC are difficult to capture in structured testing situations. Further studies on neurocognitive theories of HF-ASD are needed applying more naturalistic testing situations.

Practical implications of Studies I-II are threefold. First, a significant VIQ-PIQ discrepancy is common in children and adolescents with HF-ASD. Second, using the Auditory Attention and Response Set, Word Generation, Memory for Faces, Narrative memory, Imitating Hand Positions, Visuomotor Precision, and Design Copying subtests of the NEPSY-II as a part neuropsychological assessment of children and adolescents with HF-ASD would be beneficial to detect the most likely neurocognitive deficits in these individuals. Third, cautious interpretation of the Social perception subtests of the NEPSY-II in children and adolescents with HF-ASD is crucial. Although a child or adolescent with HF-ASD is performing adequately in these subtests, significant difficulties in emotion expression recognition and ToM abilities are still possible in more demanding everyday situations.

## 5.2 Psychiatric symptoms in HF-ASD

Higher rates of co-occurring psychiatric symptoms in the HF-ASD group (68.3%) in comparison to the TD group (11.7%), and to the prevalence rates (9.5%) in the Ford et al. (2003) study were found in Study III. These results are in line with Mattila et al. (2010) study reporting that 75% of the HF-ASD group had one or more current psychiatric disorder in a clinic-based sample evaluated with the K-SADS-PL (Kaufman et al., 1997). It should be noted that two other studies applying the K-SADS-PL reported a considerably higher rate of psychiatric symptoms (93–100%) in the HF-ASD group (Mukaddes & Fateh, 2010; Mukaddes et al., 2010). Different recruitment sources probably explain the discrepancy of the results. In turn, Mazefsky et al. (2012) found lower rates of psychiatric disorders in individuals with AS/HFA/PDD-NOS evaluated with the Autism Comorbidity Interview (ACI; Lainhart, Leyfer, & Folstein, 2003). Direct comparison between Study III and study by Mazefsky et al. (2012) is challenging due to differences between the studies regarding ASD diagnoses, age range, and gender distribution. In addition, 80% of the participants in Mazefsky et al. (2012) study were newly diagnosed with ASD. Thus, methodological discrepancies could explain the mixed results between Study III and Mazefsky et al. (2012) study.

Emotional disorder (36.7%) symptoms were significantly more frequent in the HF-ASD group compared to the TD group (5.0%) These findings are consistent with previous interview-based studies assessing psychiatric symptoms in children and adolescents with HF-ASD (Mattila et al., 2010; Mazefsky et al., 2012). Specific phobia (20%) was the most frequent co-occurring anxiety disorder in children and adolescents with HF-ASD in Study III. This finding is in line with a meta-analytic study by van Steensel et al. (2011)

reporting specific phobia being the most frequent (30%) anxiety disorder in individuals with ASD. It has been suggested that ASD-related features such as oversensitivity to sensory stimuli and idiosyncratic thoughts and interpretations of experiences probably contribute to the high rate of specific phobias in these individuals (Mayes et al., 2013).

The second and third most frequent anxiety disorders in children and adolescents with HF-ASD were GAD (10%) and OCD (8.3%). Higher rates of GAD (22-35%) and OCD (22─25%) have been reported in previous studies on individuals with HF-ASD (Green et al., 2000; Lugnegård et al., 2011; Mattila et al., 2010). There is an indication that GAD and OCD manifest with increasing age (for a meta-analysis see, van Steensel et al., 2011). Thus, the inclusion of younger participants in the present thesis might have led to lower GAD and OCD rates in comparison to studies with older participants. It should be noted that restricted and repetitive behaviors in ASD resemble OCD symptoms closely and one could argue that high rates of OCD in ASD could be just a manifestation of symptom overlap rather than two co-occurring disorders (Mazefsky et al., 2012; van Steensel et al., 2011; Zandt, Prior, & Kyrios, 2007). However, Ruzzano, Borsboom, and Geurts (2015) found in a network analysis that autism and OCD symptoms yielded two separate clusters, thus indicating that OCD symptoms were distinct behaviors from the ASD-related restricted and repetitive behaviors. In contrast to expectations, the rate of PTSD in individuals with ASD did not differ significantly from the prevalence rates of Ford et al. (2003) study. Concerning depressive symptoms, significant differences were found in depressive disorder NOS in individuals with HF-ASD in comparison to the prevalence rate (0.2%) reported by Ford et al. (2003).

The rates of ADHD/hyperkinesis were significantly more frequent in the HF-ASD (26.7%) group than the TD group (1.7%). In previous interview-based studies, higher rates of ADHD (40–67%) were found in individuals with HF-ASD (Green et al., 2000; Klin et al., 2005; Mattila et al., 2010; Mukaddes et al., 2010). The qualities of the DAWBA possibly partly explain the lower rate of ADHD symptoms in this thesis. Posserud et al. (2014) found that fewer ADHD diagnoses were assigned on the basis of DAWBA in comparison to other instruments. Another possible reason for the lower rate of ADHD in this thesis is the lack of information from the teachers regarding ADHD symptoms of the participants. This likely led to missing some of the ADHD symptoms. Thus, these results should be taken only as suggestive.

Tic disorder symptoms were significantly more common in children and adolescents with HF-ASD (18.3%) than in TD children and adolescents (1.7%) and in study (0.1%) by Ford et al. (2003). Contrary to expectations, none of the participants in the HF-ASD group had anorexia nervosa or bulimia nervosa. No studies investigating these symptoms have been conducted in children or adolescents with HF-ASD. Bölte, Özkara, and Poustka (2002) examined the body mass index (BMI) of older individuals (age range 10.1–39.9; mean age 19.7) with autism (n = 71) and AS (n = 32). In their study, none of the participants had anorexia nervosa although they found that 28% and 3% of male and female participants had lower than an expected body mass index.

To conclude, in Study III children and adolescents with HF-ASD had a significantly higher rate of psychiatric symptoms in comparison to the TD group and in comparison to the prevalence rates reported by Ford et al. (2003). Specifically, emotional disorders,

ADHD/hyperkinesis, and tic disorders were significantly more prevalent in the HF-ASD group compared to the TD group. The results of Study III highlight the importance of assessing symptoms of emotional disorders, ADHD/hyperkinesis, and tic disorders in clinical work when a child or adolescent is suspected to have an ASD or is followed-up because of ASD. This could lead to earlier identification of co-occurring psychiatric symptoms and therefore would enable planning accurate interventions. Compared to other psychiatric interviews, the DAWBA could be an especially useful assessment method for the early recognition of these symptoms in children and adolescents with HF-ASD. First, high specificity of the DAWBA in comparison to other diagnostic assessments have been reported (Posserud et al., 2014). The DAWBA being a conservative method is important when considering a child or adolescent with HF-ASD because avoiding unnecessary evaluations and wrong interventions with children with ASD is essential for diminishing the family stress. Mothers of children with ASD have more stress compared to mothers of children with developmental disabilities without autism (Estes, Munson, Dawson, Koehler, Zhou, & Abbott, 2009). Second, no extensive training is needed to conduct the DAWBA and also self-completion versions of the DAWBA are available. This enables fast and less burdensome assessment of psychiatric symptoms as part of other evaluations relating to ASD. Third, the possibility to conduct the DAWBA online is important when considering children and adolescents with HF-ASD taking into account their difficulties in social interaction.

## 5.3 Sluggish cognitive tempo in HF-ASD

The main findings of Study IV were that individuals with HF-ASD+High SCT had significantly more social difficulties and a higher rate of internalizing disorders than individuals with HF-ASD+Low SCT after statistically controlling for symptoms of ADHD-I and ADHD-HI and age. The HF-ASD+Medium SCT group had significantly more social difficulties and marginally significantly higher rate of internalizing disorders than the HF-ASD+Low SCT group when symptoms of ADHD-I and ADHD-HI and age were statistically controlled. These results are in accordance with previous research reporting associations between SCT symptoms and social impairment, as well as associations between SCT and internalizing symptoms in children with ADHD and TD children (Becker, 2014; Becker et al., 2016b; Becker & Langberg, 2013; Becker, Marshall, & McBurnett, 2014; Lee et al., 2014; Servera et al., 2016; Willcutt et al., 2014).

In contrast to expectations, no significant group effect was found regarding academic functioning after statistically controlling for symptoms of ADHD-I and ADHD-HI. In previous studies, results concerning these associations have been inconsistent (Becker & Langberg, 2013; Servera et al., 2016; Watabe, Owens, Evans, & Brandt, 2014; Willcutt et al., 2014). The results of Study IV are consistent with Becker and Langberg (2013) and Watabe et al. (2014) who found no significant associations between SCT and parent-rated academic impairment. In contrast, Willcutt et al. (2014) and Servera et al. (2016) reported significant associations between SCT and parent-rated academic functioning after controlling for symptoms of ADHD-I and/or ADHD-HI. Comparisons between the studies

are difficult due to the different assessment methods. It is also noteworthy that small sample size (HF-ASD+High SCT group $n$ = 13; HF-ASD+Medium group $n$ = 18; HF-ASD+Low SCT group $n$ = 12) in Study IV on academic functioning is one possible reason for the non-significant results.

With respect to processing speed, non-significant group differences were found. The results concerning processing speed and symptoms of SCT have been contradictory, and only initial support for the association of slow processing speed and symptoms of SCT was reported in a meta-analysis and a critical review (Becker et al., 2016a). The non-significant results in this study might be explainable by the overall small association between symptoms of SCT and slow processing speed (for a meta-analysis and a critical review see, Becker et al., 2016a) and small sample size.

Overall the results of Study IV suggest that SCT is a separate disorder from ADHD. Similar relations between SCT and functional impairments emerged in HF-ASD beyond symptoms of ADHD as also has been reported in other clinical conditions. In Study IV 30% of children and adolescents with HF-ASD belonged to the HF-ASD+High SCT group indicating a high rate of SCT symptoms in individuals with HF-ASD.

An interesting question is why children and adolescents with HF-ASD seem to be likely to have SCT symptoms. At present, the underlying mechanisms of SCT are unknown, and there is no theory on SCT. Genetic, neurobiological, and environmental factors could contribute to the high rate of SCT in individuals with HF-ASD. First, SCT has been found to be moderately heritable and over half of SCT genetic contribution is shared with ADHD (Moruzzi, Rijsdijk, & Battaglia, 2014). It is likely that genetic variables are related to SCT symptoms in ASD since ASD is also highly heritable and there is a shared genetic contribution in ASD and ADHD (Rommelse, Franke, Geurts, Hartman, & Buitelaar, 2010). Second, neurobiological factors may influence the SCT symptoms in ASD. SCT symptoms have been found to be related with the left superior parietal lobe hypoactivity (Fassbender, Krafft, & Schweitzer, 2015), which belongs to the orienting attentional network (Petersen & Posner, 2012). In ASD, impairments in the orienting attentional network have been found in many studies (Keehn, Müller, & Townsend, 2013). It is possible that individuals with HF-ASD and with impairments in the superior parietal lobe are at an elevated risk of having symptoms of SCT. Finally, environmental factors could also be contributing to symptoms of SCT in ASD since in ADHD it has been found that the environmental factors influence the symptoms of SCT considerably (Moruzzi et al., 2014). At the moment, the specific environmental variables contributing to SCT symptoms are not known (Becker et al., 2016a). With respect to theoretical accounts on SCT, a number of possibilities concerning the causes of SCT symptoms have been proposed by Barkley (2014) although no specific theory on SCT has not yet been formulated. These possibilities include deficits in arousal, pathological mind wandering, difficulties in motivation, ruminative/obsessional impairment or difficulties in vigilance. Studies on SCT do not support deficits in arousal or difficulties in motivation as likely causes for SCT according to Barkley (2014).

Study IV indicates that symptoms of SCT are separate from symptoms of ADHD since similar associations in HF-ASD were found as have been reported in other clinical conditions (Becker et al., 2016b; 2016; Garner et al., 2010; Garner et al., 2013).

Discussion concerning whether SCT is a distinct disorder or a transdiagnostic construct is going on in the SCT literature. Transdiagnostic construct is used to refer to traits, processes or symptoms (e.g., deficits in emotion regulation) that occur across separate diagnoses, and that may predispose an individual to disorder and/or that could maintain a disorder (Becker, 2014; Becker, Ciesielski et al., 2016d; Egan, Wade, & Shafran, 2011). No SCT diagnose is defined in the DSM-5 (American Psychiatric Association, 2013). According to Barkley (2014), SCT may be a distinct psychiatric disorder while Becker et al. (Becker, 2014; Becker et al., 2016a; Becker et al., 2016d), have suggested that SCT can be better conceptualized as a transdiagnostic construct. Study IV extends the previous research by indicating that SCT symptoms occur at high rates also in HF-ASD. Therefore, Study IV gives preliminary support for the suggestion that SCT may be a transdiagnostic set of symptoms rather than a separate disorder.

Taken together, children and adolescents with HF-ASD and with high levels of symptoms of SCT had significantly more pronounced social difficulties and a higher rate of internalizing psychiatric symptoms in comparison to those with low levels of SCT symptoms. The findings of Study IV indicate that children and adolescents with HF-ASD and with high levels of SCT symptoms may have a higher risk for functional impairments compared to children and adolescents with HF-ASD with low levels of SCT. Therefore, recognizing SCT symptoms in ASD as early as possible would be important to assist planning follow-up and preventive support.

## 5.4 Study limitations

Studies I-IV have a number of strengths such as homogeneity of the sample (ASD diagnosis, age-range and intellectual functioning), comprehensive assessment of neurocognitive functions and psychiatric symptoms applying clinically relevant methods and gaining information concerning less studied areas such as neurocognitive functioning in adolescents with HF-ASD and also gaining information concerning areas studied first time such as SCT in HF-ASD. Alongside with the strengths of the Studies I-IV, this thesis has some limitations. First, Studies I-IV were clinic-based studies, and all children and adolescents with HF-ASD were recruited from units specialized in the ASD assessment. Therefore, the results of this thesis may not be generalizable to community-based samples. Second, although the sample size was reasonable in this thesis in comparison to previous studies assessing neurocognitive functioning and psychiatric symptoms in HF-ASD, the sample size was still limited with respect to statistical power (Cohen, 1988). Thus, possibly some group differences were not discovered because of the small number of participants.

Third, limitations concerning the assessment methods should be noted. Administration and scoring of neuropsychological subtests are challenging tasks and many factors can affect the results such as the professional-participant interaction, and the training and experience of the professional in neuropsychological assessment. Thus, these factors may have influenced the results of Studies I-IV. It is important to note, however, that in this study, the professionals conducting the neuropsychological subtests were

mainly neuropsychologists and psychologists who had experience in test administration and scoring. Training and supervision was provided for the less experienced professionals. Finally, limitations of the assessment of psychiatric symptoms and SCT in Studies III and IV should be noted. Information on psychiatric symptoms was collected solely from the parents and not from children, adolescents, or from school in Studies III-IV. Therefore, the rates of psychiatric symptoms in Studies III-IV may be attenuated due to the use of a single source of information instead of multiple sources. Additionally, parent and family-related (i.e. parent-child relationship) factors, the observability of symptoms to parents as well as parent's willingness to report the symptoms may have affected the results (Karver, 2006; Treutler & Epkins, 2003). However, a strength in the assessment of psychiatric symptoms in Studies III-IV is that information of psychiatric symptoms was gained by combining parent-ratings and parent's open descriptions of their child's or adolescent's behavior in the DAWBA with experienced child psychiatris's evaluation of this information. Another limitation relating to the assessment methods is that SCT symptoms were not assessed with a comprehensive SCT rating scale (Burns & Lee, 2011; McBurnett & Pfiffner, 2005; Penny et al., 2009). Instead, two FTF items (Kadesjö et al., 2004; Korkman et al., 2005) were used to evaluate SCT symptoms in HF-ASD. Further studies on SCT in HF-ASD applying comprehensive SCT rating scale for the assessment of SCT are needed.

## 5.5 Conclusions and clinical implications

The present theses investigated neurocognitive functioning and psychiatric symptoms in children and adolescents with HF-ASD. Since ASD is a highly heterogeneous disorder, this thesis aimed at diminishing the heterogeneity by focusing solely on children and adolescents with one specific ASD diagnosis, that is, Asperger syndrome. Concerning the neurocognitive level, the present thesis supports most the EF and WCC accounts of ASD instead the ToM deficits account. It should be noted that at group-level, the neurocognitive deficits were mild in children and adolescents with HF-ASD. Strengths in verbal reasoning skills and weaknesses in attention and EFs, facial recognition memory, and visuomotor functions were found in the present thesis in children and adolescents with HF-ASD. High heterogeneity regarding neurocognitive functioning in these individuals was noted despite the fact that this thesis focused on a narrowly defined group of children and adolescents with ASD. The clinical implications of the neurocognitive studies (Study I and Study II) of the present thesis are as follows 1) neuropsychological assessment of these individuals is important due to the high heterogeneity of ASD, 2) cautious interpretation of performance regarding the Social perception subtests of the NEPSY-II is crucial concerning children and adolescents with HF-ASD, and 3) the use of standardized rating scales to obtain information from everyday difficulties is important.

Concerning the psychiatric level, high co-occurrence of psychiatric symptoms in children and adolescents with HF-ASD was found in line with previous studies. The clinical implications of Study III are as follows 1) screening and/or assessment of the symptoms of anxiety and depression, ADHD, and tic disorders is essential in children and

adolescents with HF-ASD during initial ASD evaluation or follow-up, and 2) the DAWBA interview could be a useful interview for the initial assessment of psychiatric symptoms in individuals with HF-ASD.

Finally, in Study IV children and adolescents with HF-ASD and with high level of symptoms of SCT had the most severe social difficulties and higher rates of symptoms of anxiety and depression compared to children and adolescents with HF-ASD with low levels of SCT. The clinical implication of Study IV is that assessing SCT symptoms in individuals with HF-ASD is important.

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